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A TREATISE

ON

AUSCULTATION AND PERCUSSION.

A TREATISE

ON

AUSCULTATION

AND

PERCUSSION,

BY

DR. JOSEPH SKODA.

TRANSLATED FROM THE FOURTH EDITION,

BY

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TRANSLATOR'S PREFACE.

THE author of the following Treatise has long held a high position as a clinical teacher in Germany. The novelty of the opinions which he professes on many points connected with auscultation and percussion, has excited the attention and evoked the criticisms of late writers on thoracic diseases, both here and in France; and his name has in consequence become familiar to the student of medicine in this country. His doctrines, as well as those of the modern German school, upon the same subjects, have been prominently brought before the profession, through the excellent work of Dr. Davies.

I have undertaken this translation of Skoda's work, from a conviction that a more general diffusion of its contents can scarcely fail to contribute to the advancement of the special methods of diagnosis of which he treats. I have certainly formed a very high opinion of its merits; I would almost venture to assert, that it is the most valuable treatise on auscultation which has appeared since the time of Laennec: it contains a philosophic investigation of many of the leading facts connected with the study of auscultation and percussion, and is something more than a mere manual. The reader feels that he is in the hands of a deep and an original thinker, and of a most cautious

and a clear-seeing observer—of one, indeed, who has had vast experience, and who manifests the rare quality of a will and a power to subject theory to the results of practice and experiment. Skoda will not be found to offer vague opinions, or to indulge in flights of fancy; he comes before us "les pièces à la main"; the grounds on which his doctrines rest are plainly exposed, so that there is opportunity for those who reject them to show, either that his experiments or his data are faulty, or that his deductions are not warranted by his premises; happily, his experiments are generally of such a character as to permit of being readily repeated by any one possessed of an ordinary degree of skill.

It is certainly no slight testimony in favour of the value of Skoda's opinions, that they should, after having been overlooked, or ignored as theoretical and imaginative, during the long period which has elapsed since the publication of the first edition of his Treatise, have quietly worked their way, and, as it were, forced themselves upon the attention of medical observers; in this respect, at least, his work offers a marked contrast to many modern ephemeridæ.* I might also add, that those who are best acquainted with his labours, appear also to be his warmest admirers.

No one, I should imagine, can have paid much attention to the subject of physical examination of diseases of the thorax, without feeling that a vast deal of difficulty and obscurity is still attached to it (more, indeed, than our pride willingly permits us to confess), and that we have much to learn, and perhaps much to unlearn, before we can hope for anything like perfection in our diagnosis

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* "Was glänzt, ist für den Augenblich geboren,
Das Aechte bleibt der Nachwelt unverloren."

GÖTHE.

of these diseases. How often has the most skilful and practised observer to do penance for his errors, before facts revealed by the knife of the anatomist! Is it not reasonable to suppose, that our *principia* must be somewhere faulty?

Skoda would seem to have felt these doubts and difficulties, and to have therefore entered into a critical examination of some of those leading doctrines of auscultation, which have hitherto, since Laennec wrote, received almost universal assent. Laennec's vast authority would naturally tend to immortalize even error, when it proceeded from himself. From certain of the doctrines of this great man, Skoda dissents; he gives his reasons for so doing: and I can scarcely believe that it is possible for any unprejudiced person to peruse the following pages, without arriving at the conviction, that many of Laennec's opinions, and therefore, generally speaking, the opinions of the present day, require modification, and that some of them must be rejected altogether; even those who refuse the theories offered by Skoda in their place, must admit this much. If our author has done no more than clear away errors which encumber progress, he has surely done much—no superstructure can be secure, whilst its basis rests on untruth.

I do not intend to enter here into any examination of the particular doctrines which Skoda holds; they must be studied *in extenso*, before they can be fairly appreciated. I am sure that a partial consideration of them has led more than one of his critics, and consequently those readers who gain their knowledge of them through such critics, into an erroneous interpretation of their real meaning. If obscurity attaches itself to isolated portions of an author's reasonings on any particular subject (and, in some rare instances, this fault may perhaps be objected to Skoda), it is evident that the reader should endeavour to

seek his real meaning in a fair consideration of the whole of the argument, and not by partial views of it.

In making this remark, I would particularly refer to Skoda's explanation of bronchophony by the theory of consonance—a subject in connexion with which his name is most generally associated in this country. I will here give a short sketch of this theory, and of the arguments by which Skoda supports it: for its full comprehension, I must beg the reader to study the author's own account of it.

. . . . The voice passes into the parenchyma of the lungs through the medium of the air in the trachea and the bronchial tubes, and is not propagated along their walls; it traverses healthy, as readily as it does hepatized lung, and even somewhat more readily: consequently, bronchophony does not depend upon an increase of the sound-conducting power of consolidated pulmonary tissue; moreover, when the lung is consolidated, the thoracic voice increases and diminishes in force, without any concurrent change taking place in the condition of the lung: this variation in its strength evidently results from the circumstance of the bronchial tubes being at one moment blocked up by mucus, etc., and at another freed therefrom by the cough and expectoration, etc.: if the bronchophony depended upon conduction of sound, it would be a matter of indifference whether the tubes contained air or fluids. It must not be forgotten, that, according to the ordinary laws of reflection of sound, the more solid the parenchyma the more difficult does the passage of the sound from the air into it become.

That the air in the mouth and nasal cavities consonates with sounds formed in the larynx, is proved by the fact of the changes which the voice undergoes through opening and closing the mouth and nose, whilst the condition of

the larynx remains unaltered; just in the same way does the air in the trachea and bronchial tubes consonate with the laryngeal sounds. Now, air consonates only in a confined space, and the force of the consonance depends upon the form and size of the space, and upon the nature of the walls forming it: the more solid the walls, the more completely will the sound be reflected, and the more forcible the consonance. The cause of the loud voice produced by a speaking-trumpet is well known. But the air will consonate with certain sounds only; in the trachea and bronchial tubes, it becomes consonant with the laryngeal voice, in so far as their walls have a like or an analogous character to the walls of the larynx, of the mouth, and of the nose. Within the cartilaginous walls of the trachea and the bronchial trunks, the voice consonates nearly as forcibly as in the larynx; but as the bronchial tubes divide in the lungs, they lose their cartilaginous character, becoming at last merely membranous in structure, and therefore very ill-adapted for consonance; when, therefore, the consonance is increased in these latter tubes, we may be sure, either that the membrane forming them has become very dense or cartilaginous, or that the tissue around them is condensed and deprived of air, whereby the sound-reflecting power of the tubes is increased. Of course the communication between the air in the tubes and the air in the larynx must be uninterrupted.

The walls of a confined space frequently vibrate in unison with sounds excited within it, as do those of an organ-pipe, or of a speaking-trumpet. The larynx vibrates with every sound, and its vibrations are perceptible at a considerable distance from their point of origin; so, also, must the walls of the bronchial tubes, which are distributed through the parenchyma of the lungs, vibrate when the voice consonates within them; and the vibrations thus ex-

cited will extend to the surface of the thorax, passing through several inches of thick fleshy parts, or of fluids, and manifest themselves there as the consonating sounds of the bronchial tubes. . . . .

Such is a general account of Skoda's theory of the cause of the bronchophonic voice. The critical manner in which it has been brought before the profession, has certainly not much aided in its propagation; and I suppose, with the great majority of physicians amongst us the theory in question is looked upon as little more than a piece of German mysticism: the reason of this, in my opinion, is, that the subject has come before them *in partibus*, and not as a whole. Whatever may be the ultimate fate of this theory, one thing may be very fairly predicted to result from Skoda's enunciation of it, and from a consideration of the experiments and reasonings on which he bases it, and that is, a recognition of the fact, that Laennec's explanation of bronchophony, by the increased sound-conducting power of consolidated pulmonary tissue, is not true, at least, in every case.

It is scarcely possible, moreover, that the remarkable results which have in other respects rewarded Skoda's labours, so completely opposed as they often are to the opinions which men, since the time of Laennec, have held as undeniable, should fail in other hands to produce fresh additions to our knowledge; what, for instance, is more opposed to our preconceived notions respecting the conducting power of pulmonary tissue, than the statement, that sound passes more readily, and is heard to a greater distance, through healthy than through hepatized tissue? What, again, can appear more contradictory than an assertion of this kind: that a cavity, such as the stomach or the thorax, when its walls are tensely inflated with air, yields a dull, non-tympanitic percussion-sound, but that

when its walls are lax, the sound becomes distinctly tympanitic?

The work is throughout of a suggestive character, and in such a point of view cannot, I believe, fail to be of service to the thoughtful student; it certainly is not to be regarded merely as a hand-book of details: in this respect, it might perhaps be considered as somewhat defective, for there are certain subjects in it which have been but lightly touched upon, and are meagre in description. I might instance the chapter on percussion of the abdominal organs, and the account of aneurisms. I once had an idea of attempting to fill up what I believed was wanting, with the hope of rendering the book more complete for the hands of the student; but upon reflection, I have thought it better that the work should retain its original form; its deficiencies may be readily supplied from numerous other sources, and further additions of the kind referred to, might perhaps only detract from the character and peculiar value of it.

Neither have I thought it advisable to omit any of the controversial portions of the work; these may here and there seem over long, perhaps even wearisome, but to have passed them over would hardly have been doing justice to their author. The general tenour of them is undoubtedly excellent: Skoda's argument is straightforward and good, logical, consequent, and to the point, and he has on more than one occasion left the battle field strewn with the *disjecta membra* of many of those authorities whose opinions he has assailed.

Skoda has also done excellent service by attempting to reduce to their real practical value, the results of auscultation and percussion, affixing to each sign its true signification, and by drawing distinction between those signs which have, and those which have not, special indications; in truth, throughout, this seems to have been one of the

main objects which he keeps constantly before him. He certainly cannot be accused of drawing pale and sickly conclusions from his observations: whatever is not clear and manifest on the face of it, is at once classed by him as indeterminate and as incapable of service in the field of medical observation, and as such to be rejected from our catalogue of signs and symptoms; that is, in so far as drawing practical conclusions from them is concerned.

The crime of endless subdivision, which is often charged to the account of the German school, cannot be laid at his door; rather, I fear, will he be accused of a certain scepticism, of a barrenness of faith, which has brought him to give too little credence to the value of those minutiæ of detail, in which men now-a-days are wont so frequently to indulge. It is really strange to find an observer, an acknowledged master of his subject, unrivalled in powers of diagnosis, as they who have witnessed his skill tell us that he is; it is strange, I say, to find such a man on almost every occasion warning his readers, when they have in any particular case obtained all the information which auscultation can afford them, still, before they conclude their diagnosis, to pause, and to lay hand upon every other aid which other sources can supply; as if he thought and felt that the observer may be falling into error, even then when he thinks himself most secure. Surely the inference from this is manifest, viz.: that men are wont to force from the physical examination of diseases of the thorax, consequences which are not legitimately produced by it.

The truth of the inference I most thoroughly believe, and if I might venture, out of my own experience, to offer advice to those commencing the study of auscultation, I would strongly warn them from putting their confidence in that kind of teaching which boasts that auscultation

can demonstrate the hidden workings of the thoracic organs, with something of the same degree of certainty as if they were the direct objects of the vision. I would rather say, approach the subject with a caution somewhat akin to fear and trembling; fix your faith as clearly as you please on clear, broad, and manifest positions, whenever you are certain that you have gained them, but play the sceptic's part with doubtful signs; assign them whatever legitimate value may fairly attach to them, but never let them *rule your judgment*, for you will find them only too apt to do so when the diagnosis is difficult. I would not venture to offer such advice, if it had been drawn from the experience of my own errors; but I take for the grounds of my warning the mistakes of far more skilful observers than myself. I cannot refrain from noticing the subject, when I see the unhesitating manner in which conclusions are so frequently drawn from these indefinite signs, and the practical applications which men found upon them and teach others to found upon them.

If I am asked to what I refer when speaking of doubtful signs, I answer, every sign which does not present itself clearly and manifestly to the sense, as the acknowledged exponent among ordinarily skilled observers, of certain special conditions of the internal organs.

The recognition of pathognomonic signs in auscultation by authors, especially of the French school, has been, I believe, one great source of the mischief I refer to. The classification of auscultatory sounds according to the stages and processes of diseases which produce them, is manifestly erroneous, and cannot fail to leave faulty impressions on the minds of those who put their trust in it: as an example of what I mean, I would point to the flatulent nosological vocabularies of writers like M. Fournet; this author wishes his readers to believe, that every diseased condition of the lungs has its appropriate sign, and

every part of the lungs its peculiar râle, and he tells with the most perfect gravity of râles such as these:

"Râle humide à bulles continues de la congestion sanguine.

"Râle sous-crépitant de l'œdème pulmonaire.

"Râle sous-crépitant du catarrhe pulmonaire aigu capillaire.

"Râle sous-crépitant ou crépitant de retour de la pneumonie.

"Râle crépitant primitif de la pneumonie."

Now, all this is evidently a misconception: for, as Skoda justly observes, there is assuredly no distinct râle peculiar to any of these morbid states of the lungs to which Fournet alludes. That the varieties of râles described by him may have a real existence, Skoda does not deny; he says, indeed, that many more might be added to them; but as external manifestations of the special pathological conditions of the lungs which they pretend to indicate, he utterly rejects them.

Perhaps no one thing has more tended to impede the progress of auscultation, than the vagueness and diversity, I might say the repulsiveness, of the terms employed by different authors to indicate its phenomena: such is the confusion thereby introduced into medical literature, that I feel convinced that the idea conveyed to the mind of the reader, is frequently different from that intended by the writer. How can it be otherwise so long as authors use the same term in different senses? Ask a dozen physicians what they mean by muco-crepitant râle, and I very much doubt whether any two will give the same account of it.

The introduction into our language of a plain practical nomenclature of auscultation is much required, and would be a great boon conferred upon the science. Skoda's

nomenclature is eminently plain and to the purpose, but unfortunately it is in part based upon his theory of consonance: he speaks, for instance, of consonating râles: so that its adoption can, I fear, hardly be hoped for until such time, at least, as his theory has met with more general approbation.

I cannot, however, refrain from calling attention to the simplicity and practical value of his method, based as it is on strictly philosophical principles. The respiratory murmurs and the râles, according to his division of them, fall into two distinct categories, viz. râles and murmurs which are the indicators of certain conditions of the organs of respiration, and râles and murmurs which indicate nothing special; the latter he calls indeterminate, and for practical purposes sets them aside, as being valueless in a diagnostic point of view. The râles he divides as follows:

Vesicular râles.

Consonating râles.

Dry crepitating râle with large bubbles.

Râles accompanied by metallic tinkling and amphoric echo.

Indeterminate râles.

I have already mentioned the value to be attached to the latter sort, the indeterminate.

The vesicular râle arises in the finer bronchial tubes and air-cells, and indicates the presence there of mucus, blood, serum, etc., and the passage of air into them, excluding therefore all those diseased conditions, by which the entrance of air is prevented; but it indicates nothing more than this.

Consonating râles occur when that condition of the lung is present which gives rise to bronchial breathing and bronchophony, and they have therefore the same indications.

The dry crepitating râle of Laennec, Skoda retains, but he looks upon it as of little value. He attributes its cause to the distension of the walls of the air-cells, bronchial tubes, and cavities during inspiration, when they have lost their natural contractility, and simply collapse during expiration.

The nature of the râles accompanied by metallic tinkling and amphoric echo is sufficiently indicated by their title.

All these râles have more or less a character of moistness. The dry sounds heard during respiration, etc., are placed apart from them, and are designated by Skoda as hissings, whistlings, and snorings; they arise for the most part as follows: the snorings in the large bronchial tubes, the whistlings in tubes of a lesser diameter, and the hissings in the finest portions of the air-passages; they are produced by the passage of the air through the tubes when narrowed, etc. For their value as diagnostic signs, I refer the reader to Skoda's account of them.

Equally simple and philosophic, is his division of the murmurs heard during respiration:

Vesicular breathing.

Bronchial breathing.

Amphoric echo and metallic tinkling.

Indeterminate respiratory murmurs.

His division of the thoracic voice is founded on the same principles; it is carried only so far as it has a practical bearing. Laennec's bronchophony and pectoriloquy are rejected because they represent one and the same phenomenon, and I believe justly so. Is there one man of experience in fifty who can deny that the sign called pectoriloquy has not been at some time or other a stumbling-block and a source of error to him; that it has not.

in fact, been the cause of his diagnosing the existence of a pulmonary cavity, when none existed?*

Œgophony is also excluded as being a sign of no especial value, it is occasionally heard in connexion with the consonating voice, but has no necessary relation with the existence of fluid in the pleura.

I have dwelt somewhat long upon this subject; but I have done so from conviction that an adoption of Skoda's simple and philosophic method of dealing with it, or of some similar method, would be a great source of relief to the perplexed mind of the student, and would introduce something of order into the endless confusion of terms, which hovers like an incubus over the study of auscultation. How is it possible that precise ideas of the value of a particular sign can fix themselves in the student's mind, when the sign is represented to him by different authors under such a variety of terms? Here, for instance, are a few of those by which the "râle crépitant humide" of Laennec is known amongst us: moist crepitous rhonchus, crepitation, crepitating râle, crepitant rhonchus, crepitant râle, minute crepitations, crackling of pneumonia, small crepitations, vesicular râle, râle sous-crépitant du catarrhe pulmonaire aigu capillaire. In the present state of our nomenclature, again, how can the reader, when he meets with expressions like the follow-

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* Skoda tells us, that percussion and auscultation afford very few certain signs of the presence of a cavity, viz., amphoric echo and metallic tinkling, and the cracked-pot sound, and that these signs are not frequently met with, because the conditions necessary for their production occur exceptionally, and not as the rule; so that in the majority of cases of phthisis, it is by inference only that we can foretell the presence of a cavity; experience teaching us, that tubercular masses rarely exist for any length of time without producing cavities.

ing, be sure that he is interpreting them according to the writer's meaning? large crepitating râle, mucous râle, bubbling râle, sub-crepitant rhonchus, muco-crépitant râle, sub-mucous rhonchus, dry mucous rhonchus, cavernous rhonchus,—for what standard have we by which to measure their true signification?*

It surely is unnecessary to waste the time of the reader by any further illustrations of the confusion which pervades this subject of nomenclature, and which taints the whole study of auscultation.

The portion of Skoda's work devoted to the phenomena presented by the organs of circulation, will be found not less valuable than his account of the phenomena presented by the organs of respiration. Dr. Davies bears testimony to its excellence in terms of the highest praise. The same caution is here displayed in drawing conclusions, and the same endeavour to reduce to their just value the signs which offer themselves to the observer; and the whole subject is treated of in a precise and methodical manner.

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* I cannot leave this subject without referring to the earliest of Dr. Latham's admirable little volumes on "Subjects connected with Clinical Medicine". He is the first English author, so far as I am aware, who has endeavoured to throw a little Saxon simplicity into the study of auscultation, and to divest it of some of its Gallic exuberances. "Auscultation," he writes in his preface, "is capable, I have thought, of being greatly simplified for practical purposes. At all events, unless it be so, it can never be successfully taught; the knowledge derived from it must be confined to a few physicians of hospitals, and the profession at large can never expect much benefit from it."

Further on, he tells the student to "guard himself against over-refinement", in studying the facts of auscultation.

The following is a short summary of some of Skoda's opinions respecting the sounds, the murmurs, and the impulse of the heart.

The *impulse* of the heart depends upon a variety of causes; the force which moves Segner's wheel (better known in this country as Barker's mill) is one: this is the theory as it is called of Gutbrod;* but another cause is the lengthening of the aorta and pulmonary artery, which takes place when the blood is forced into them at each systole of the heart; something of the impulse may also be attributed to the change of form, and the rigidity which the heart undergoes during its contraction. The contraction of the papillary muscles takes no part in its production. All the causes of the impulse are not yet known.

No exact conclusion as to the condition of the heart can be drawn from the force of its impulse, so many are the disturbing causes by which it is modified: for instance, a ventricle hypertrophied and dilated is peculiarly fitted to produce a strong impulse, but it will not do so unless the heart's action be rapid and complete; and so again, the beat of an hypertrophied heart may at one moment cause violent concussion of the thoracic walls, and at another be almost imperceptible.

It is difficult to determine the *direction* which the heart takes during its systole. The contractions of the right and left ventricles tend to force the heart in different directions; its actual motion is in the diagonal of the two forces. The abnormal positions of neighbouring organs also tend to give the heart either an horizontal or a vertical position. The statement made by Laennec and others,

* Some of Skoda's critics have led their readers to believe, that he adopts this theory, as the *only* cause of the heart's impulse.

that the impulse is felt on the left side when the left ventricle is hypertrophied, and behind the sternum when the right ventricle is hypertrophied, is incorrect.

The impulse of the heart offers three different degrees of force: viz., first, when it does not shake the head of the auscultator; secondly, when it imparts a concussion to the head, but does not raise the thoracic walls; and thirdly, when it both imparts a concussion and raises the walls. The first degree, as a sign, is of little value, for its existence is compatible as well with an hypertrophied and dilated as with a normal heart; the second degree is also indefinite, for it may be occasioned by a healthy heart, or may indicate hypertrophy of one or both sides of the heart; the third degree requires for its production hypertrophy and dilatation of both ventricles. The impulse of a healthy heart is not felt over more than one, or at most, two intercostal spaces.

The aorta, when enlarged, will communicate an impulse to the thorax, where it comes in contact with its walls; the pulsations of the pulmonary artery may be felt when a consolidated portion of lung (either from tubercle or pneumonia) intervenes between it and the thorax.

The character of the heart's sounds varies much; the variation depends upon many different circumstances.

In the production of the sounds of the heart, the ventricles, the aorta, and the pulmonary artery, severally contribute a share.

The *first sound* of the ventricles is, for the most part, produced by the sudden impulse of the blood against the mitral and tricuspid valves, when they are distended and oppose its reflux into the auricles; by the state of tension into which the valves are thrown; and by the impulse of the heart against the walls of the thorax. But these causes are insufficient to explain the cause in every instance.

Greater difficulties surround the explanation of the *second sound* of the ventricles; we cannot be sure that it is always formed in the ventricles, although it is often louder at the apex than at the base of the heart; it seems pretty certain that it sometimes arises in the arteries. It may occasionally be caused by the impulse of the blood against the ventricles during the diastole.

The sounds accompanying the pulsations of the arteries may be explained by the sudden tension of their coats. The second sound heard over the aorta and pulmonary artery, is produced by the impulse of the regurgitating blood upon the semilunar valves.

The *murmurs* are, for the most part, associated with changes of the heart's structure; but they are sometimes observed in the course of certain diseases, chlorosis, fevers, etc., when no appreciable alteration exists. A murmur also arises, when a rapid current of blood is directed against blood that is quiescent, or moves less rapidly, or in a contrary direction. Murmurs which cannot be attributed to organic changes of the heart, doubtless depend chiefly upon friction between the blood and the walls of the heart. The opinion that they arise through particular conditions of the blood is hypothetical. It is not true that a watery state of the blood occasions them.

Every kind of murmur heard over the ventricles may be heard in the aorta; and murmurs are often heard over the carotid and subclavian arteries, when their coats are perfectly healthy. The placental bruit, as it is called, arises, according to Kiwisch, in the epigastric artery. It is difficult to say whether these arterial murmurs are caused by friction of the blood against the walls of the arteries, or by vibrations excited through their distension.

There is no endocardial murmur, excepting the whistling, which may not be imitated by a pericardial murmur, and no pericardial which may not resemble an endocar-

dial; we have no sign by which to distinguish them, excepting this, that the internal murmurs correspond pretty correctly to the rhythm and natural sounds of the heart, the pericardial seeming rather to follow upon its movements. A friction-sound exactly coinciding with the heart's movements, and differing in no respect from a murmur formed within the pericardium, may arise from rubbing of the roughened pleural surfaces, which cover the free portion of the pericardium, either against the thoracic walls, or against the surface of the lungs.

The cause of the production of the *bruit de diable* in the jugular veins is not very clear; it does not appear to be a sign of anæmia or of spanæmia, for it may be frequently observed in the young and healthy.

Such are the outlines of the opinions which Skoda professes concerning the sounds and murmurs of the heart. Of his rules for the diagnosis, and for the interpretation of them, it is impossible to give any summary account; these rules have evidently been proved by, and are offered as the result of, his own great experience.

The second part of his Treatise contains an account of the phenomena elicited by auscultation and percussion in the different normal and abnormal conditions of the thoracic and abdominal organs, and the application of the principles laid down in the former part of the work. This part is itself a summary of facts.

Before concluding, I must beg leave to observe, concerning the translation, that I believe I can venture to assure the reader that it conveys a correct interpretation of Skoda's ideas: in a literary point of view, I fear I must crave his indulgence, for the text will be often found not to run pleasantly to the ear: somewhat of this fault must be attributed to a constant desire of following our author very closely, and of never sacrificing the sense to the

sound, *le fond pour la forme*, and somewhat perhaps to the peculiar style of the Viennese professor.

I feel pleasure in acknowledging the advice and assistance which I have received from my friend and colleague, Dr. Sieveking, while engaged upon it.

W. O. MARKHAM.

PREFACE TO THE FOURTH EDITION.

THIS edition is essentially the same as the third. A few unimportant observations have been added to the chapter on Percussion, and an alteration has been made in the division of the thoracic voice, *loud* bronchophony having been divided into *clear* and *dull*.

I have examined most of the new theories which have been propounded since 1844, respecting the causes of the impulse, the sounds, and the murmurs of the heart, but the present edition of this work was printed before I received Professor von Kiwisch's "New researches into the causes of the sounds produced in the circulating organs"; I must, therefore, here make a few remarks on the views therein contained.

Professor von Kiwisch found, that if water was forced through a caoutchouc tube, murmurs were not produced, unless the tube was of unequal bore at different parts. The murmur invariably occurred at the wider part, immediately beyond the narrow, and never at the narrow part itself; and it disappeared, whenever an obstruction was offered to the passage of the water through the wider part. Considerable

prominences on the inner surface of the tube gave rise to a murmur, but a merely roughened surface was never observed to do so.

These facts are explained by Professor von Kiwisch in the following manner: Water flowing out from a pipe, preserves to a certain distance the form of the pipe's outlet; in this way the stream, flowing from the narrow into the wider part of the tube, retains to a certain distance the form of the narrow part, provided it has met with no particular obstruction. Now, in consequence of the pressure of the air, the walls of the wider part of the tube have a tendency to adapt themselves to the narrow stream of water, but their elasticity offers a continual resistance to the pressure of the air, and accordingly an expansion of the stream takes place. Under these conditions, the tube, alternately compressed by the atmosphere, and expanded by its own elasticity, is made to vibrate, and by its vibrations a murmur is produced.

From these experiments and their interpretation, as well as from known related physiological and pathological phenomena observable in the living body, the following conclusions may, according to Professor von Kiwisch, be drawn respecting the formation of sounds in the circulating organs of man:

"The first sound of the heart is produced by the expansion of the auriculo-ventricular valves; the second sound, by the expansion of the semilunar valves. No second sound arises in the heart itself, and no first sound in the arteries. The sound which is occasionally heard accompanying the pulse in the carotid and femoral arteries, does not proceed from

these vessels, but is caused by the vibrations excited in the air of the stethoscope, and of the ear, by the beat of the arteries.

"Murmurs arising in consequence of defect of the valves of the heart, are produced in part by the vibrations of the rigid valves, but more particularly after the manner described in the experiments with caoutchouc tubes. Thus, through defective closure of the valves, several narrow openings are formed, and through these a stream of blood is forced, with a diminished diameter, into a wide cavity: the more forcible the stream, and the less perfectly the cavity be filled—*i. e.* the less the opposition to the entrance of the blood—the more powerful and the more extensive will be the vibrations excited in that cavity.

"Murmurs are produced in arteries after the manner described in the caoutchouc tubes, and never in consequence of the presence of any roughnesses on their inner surface. Pressure upon an artery causes a murmur just *beyond* the spot compressed, never at that spot; and the murmur is more prolonged, in proportion as the muscular fibre of the individual examined is lax, and his blood poor. The so-called Nun's murmur—*bruit de diable*—is invariably formed in the carotid artery, and not in the cervical veins; it is caused, in fact, by the compression of the carotid by the omohyoideus muscle. Muscular, or any other kind of pressure, will in like manner produce this murmur in other arteries, of persons whose blood is impoverished; but it never arises in veins."

It is possible that this explanation of the mode of the production of murmurs in caoutchouc tubes may

be correct; still, I do not consider it applicable to the murmurs heard in the heart and arteries.

If the diameter of an artery be diminished by external pressure, the artery does not on that account lose its contractility; and that portion of the artery which lies beyond the spot compressed will scarcely fail to contract, its contraction being in some degree solicited by the narrowed stream of blood, now flowing into it in consequence of the pressure upon the part above.

But the in-pressing stream of blood, even though it were smaller than the arterial tube, cannot make *this* emptier: on the contrary, every artery is invariably widened by the pressure of the blood flowing into it; for its diameter is proportioned to the resistance which is to be overcome, in the distant parts of the arterial system, in the capillary vessels, and the veins, and this resistance will of necessity be increased by the in-pressure of a new quantity of blood.

That, when the mitral valves are defective, the left auricle does not become emptier at each systole, but is more fully distended, and filled by the regurgitating blood; and that, when the aortic valves are defective, the blood regurgitating into the left ventricle does not tend to contract, but rather to dilate that ventricle—are facts so well known, as not to require any comment.

THE AUTHOR.

Vienna, March 1850.

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ERRATA.

P. 42, end of line 10, for "trachea", read *larynx.*
P. 110, line 14, for "intermediate", read *indeterminate.*

PART I.

DESCRIPTION OF THE PHENOMENA, OBSERVABLE BY THE AID OF PERCUSSION AND AUSCULTATION.

CHAP. I.

PERCUSSION.

It is well known, that percussion of the abdomen has long been practised, for the purpose of ascertaining the presence of air in the intestines.

Auenbrugger taught the use of percussion in diagnosis of diseases of the thorax, and thus laid the foundation of those beautiful discoveries, which have since enriched our diagnosis of the diseases of the thoracic and abdominal organs.

Auenbrugger, Corvisart, and Laennec did not use a pleximeter, but practised percussion with the fingers only. The pleximeter was first introduced by Piorry. Piorry also pointed out, that in addition to variety of character in the percussion-sound, a resistance, sensible to the finger, was offered by the organs beneath the parts percussed: and that indications, as to the condition of these organs, were derivable as well from the nature of this resistance, as from the quality of the percussion-sound.

The pleximeter renders percussion much less irksome to the patient, and the sounds more distinct; it also facilitates investigation, for we are able, by its aid, to recognize differences in sound, which are not otherwise perceptible. It enables us, moreover, to compress the thick layers of the soft parts spread over the thorax and abdomen, and to percuss even the deep-seated regions of the abdomen, which would not be practicable without this instrument.

Piorry's pleximeter is made of ivory, and there is no necessity for our using any other; it consists of a round disk, one and a half to two inches in diameter, and so thick as not to bend: to prevent its slipping, and for its more steady application, it has either a raised border two or three lines deep, or two small projecting edges of the like depth, opposite each other.

When used, the pleximeter should be closely applied to the part percussed, and lightly or firmly pressed, according to circumstances; it should be so fixed, as not to be displaced by the percussion stroke. The blow should fall upon its centre, and be made by the middle or fore-finger, or both these fingers together, bent in a half circle; care being taken not to strike the ivory with the nails. The motion should be communicated from the wrist-joint,—not from the shoulder, elbow, or finger-joint, —whereby the loudest sound will be produced, and the patient suffer the least inconvenience. When the motion is made from the elbow or shoulder-joint, it is much less abrupt, and therefore the sound produced is not so loud, and a blow, with the whole

weight of the arm in it, falls upon the patient. The motion made from the finger-joints, is seldom powerful enough to produce a clear sound.

Louis uses a caoutchouc pleximeter, four to six lines in thickness, but it does not produce so clear and well-defined a sound as the ivory pleximeter.

Many physicians object to the pleximeter, that it is inconvenient, and excites the patient's fears, and they therefore use the finger instead. The sound produced with the finger, is very nearly as clear as that with the ivory pleximeter: but whoever practises percussion extensively, will prefer the use of the pleximeter, on account of the pain, occasioned by constant percussion upon the finger. The finger must be always used whenever, through unevenness of the surface, the pleximeter cannot be well applied.

Dr. Winterlich, of Würzburg, uses an ivory disk and a small steel hammer, into the head of which a thickish layer of caoutchouc is fixed. With this instrument a louder sound is produced than by any other, and as no particular dexterity is required in managing it, any one may, without previous practice, bring out a good sound with it. According to the observations which I have made, it does not appear that this method of percussion conduces more to correct conclusions, than percussion with the fingers. (Schmidt's *Jahrbücher*, 1841, 3 B., 2 H.)

Dr. J. Burne makes use of a disk of thick leather as his pleximeter, and a steel hammer; the head of the hammer holds a firm cylinder of leather, which projects about half an inch; the disk of leather being fixed by screws on a movable steel ring, to which

a handle is attached. It is unnecessary to show that such an instrument has no distinct object. The pleximeter of Dr. Aldis, and the modification of Piorry's by Mailliot Léon, are still more useless. (Vide Canstatt's *Jahresbericht*, 1843, p. 332.)

THE PERCUSSION-SOUND.

Sound is propagated, according to the same laws, through organic as through inorganic matter, through living as through dead bodies; but the present state of our knowledge of these laws, does not enable us to explain satisfactorily all the differences of sound which we meet with, in percussion of the thorax and abdomen; for this purpose further researches are necessary: we must first determine every possible variety of percussion-sound, and ascertain the conditions on which each variety depends, and then endeavour to reconcile our observations with the well-ascertained laws of sound. It is evident, that for the solution of this question, a vast number of experiments must be made, upon persons both in health and in disease, and on the body after death.

Varieties of the percussion-sound, and the conditions on which they depend.

With the exception of distended membranes and chords, all the soft tissues not containing air, as well as fluids, yield, when percussed, a completely dull and scarcely audible sound, which may be exemplified by percussing the thigh. There is no difference in the percussion-sound by which we can distinguish be-

tween organs not containing air, such as the liver, the spleen, the kidneys, hepatized lung, or lung completely deprived of air by compression, and fluids: a hard liver yields the same sound as a soft liver, a hard spleen as a soft spleen,—except when these organs contain bony or chalky materials,—and blood the same sound as pus, water, etc.

We may readily convince ourselves of the fact, by placing these different organs on a non-resonant support, and percussing them one after the other, either with or without a pleximeter; fluids, similarly supported, and in sufficient quantity, may also be percussed by aid of a pleximeter, carefully applied to their surface.

The sound, thus obtained from these bodies, is scarcely audible, has no tone (Klang), no distinct pitch, and no timbre, etc.

Bones and cartilages, when immediately percussed, yield a peculiar sound, but when covered by soft tissues, the sound they yield is less distinct, and altogether disappears, if the tissues are tolerably thick.

Every sound, produced by percussion of the thorax or abdomen, which differs in character, from the percussion sound of the thigh, or of bone, evidences the presence of air, or other gaseous bodies, in the parts beneath.*

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* Dr. C. J. B. Williams, in his Lectures on Diseases of the Chest, endeavours to prove, that the percussion-sound is not produced by the air within the thorax, but by the thoracic walls themselves. He explains the variations in the percussion-sound.

It may be proved by experiments on the dead body, that the soft parts of the thorax and abdomen, must be made highly tense, to produce any other sound, than the one peculiar to all soft parts.

The sound of the ribs is rarely heard, except in thin individuals, but that of the sternum, and the collar-bone, somewhat more frequently. The liver, the spleen, the heart, the kidneys, blood, water, etc., which yield, to immediate percussion, a completely dull sound, or, what is equivalent to it, no sound at all, likewise produce no sound when the parts, under which they lie in the thorax and abdomen, are percussed. The walls of the stomach, and intestines, must be in a high state of tension, to yield sound when percussed, and the same remark is true of the parenchyma of the lungs.

The different sounds, which percussion produces over the regions of the liver, the spleen, the heart, the lungs, and the stomach, do not depend upon any peculiarities in these organs, but upon variations in the quantity, distribution, and tension of the air,

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by supposing that the vibrations of the walls of the thorax, are not interfered with, by the air and healthy lung-parenchyma, but that they are impeded, or altogether destroyed, by infiltrations into the lung-tissue, by pleuritic effusions, etc., by the heart, liver, etc., and are also altered by variations in the thickness and tension of the thoracic walls. I cannot admit these views. Different sounds are heard at different parts of the thorax, and the thorax does not sound as a whole. If the thoracic walls themselves produced the sounds, they would yield the same sounds when detached; but individual parts of the thorax, when thus separated, yield the dull percussion-sound of the thigh.

present in the regions in which they lie, and upon the force of the percussion stroke.

There is no such thing as a liver-, spleen-, heart-, lung-, or stomach-sound: the sound over the lung may, under certain circumstances, be exactly similar to the sound produced by percussion over the liver.

The various percussion sounds of the thorax and abdomen, cannot be arranged together in one class, comprising every degree of sound; it is necessary to distinguish four principal varieties; the extremes of these varieties, between which there are numerous gradations, may be represented by the following terms:—*

1. Full—Empty.

---

* Laennec and Piorry divided the sounds of percussion into two classes, the clear and the dull. The latter, however, introduced numerous gradations of these divisions, and termed them: thigh-sound, liver-sound, spleen-sound, kidney-sound, intestine-sound, stomach-sound; and besides these, he describes a bone-sound, a water-sound, an hydatid-sound, and a broken-pot-sound. As we have already said, the thigh, the liver, the heart, the spleen, the kidneys, etc., all yield a similar sound, so that we cannot speak of degrees of sound in them. The lungs, as a rule, give a different sound from the stomach; but the stomach-sound may exactly resemble that of the lungs. The sound of the lungs, of the intestines, and of the stomach, is very different, and, as we have already shown, the difference depends upon the air contained in them, and is never produced by the parenchyma of the lungs, or the coats of the stomach and intestines. Piorry's division of sound is therefore not practical, and altogether unfounded in fact; it has caused many physicians, who have not had an opportunity of investigating the principles of percussion, to mistake his views entirely.

2. Clear—Dull.
3. Tympanitic—Non-tympanitic.
4. High—Low.

A full percussion-sound may be clear or dull, tympanitic or non-tympanitic, high or low; and the same is true, as regards an empty sound.

First Class:—*Percussion-sounds. Full—Empty.*—We do not judge of the size of a resonant body by the strength of the sound which strikes upon the ear; the slightest vibration of a large bell, tells of its magnitude; the loudest ringing of a little bell, misleads no one as to its smallness: neither do we judge of the dimensions of bodies, from the pitch of their sounds.

There is no good general term, to designate that quality of sounds, which characterizes the size of bodies. I believe that in singing, and instrumental music, the word full, or full-toned, or sonorous, is used; I shall therefore borrow the expression, in speaking of the percussion-sound. When any one percusses, with equal force, different parts of the thorax and abdomen, he will find that in some places, the sound appears more persistent, and, as it were, spread over a larger surface, than it does in others; the first kind of sound, I call the full; the second, the less full, or empty percussion-sound.

A cavity, superficially situated in the lungs, of moderate size, and surrounded by thickened parenchyma, yields a very distinct percussion-sound, but of an empty character. The stomach distended with air, gives a full—the small intestines, an empty sound. We do not, however, obtain similar full

sounds in different individuals, even though the superficial extent of their lungs, and the amount of air contained in them, be exactly alike; for the sounds are modified by the state of the walls of the thorax. The more yielding the walls, the greater is the effect of the stroke upon the contained air, and the more extensive the vibrations of the air thereby produced; but when the walls of the thorax are unyielding, it is difficult to obtain any sound, even from the parts which lie most superficially beneath them.

Let any one percuss portions of lung or intestines, when taken out of the body, and he will soon convince himself, that it is impossible to determine with accuracy the size of the lungs, or the width of the intestines, by the aid of the varying full sound. It is only when the most marked differences exist, between the full and the empty sound, that any certain conclusions can be drawn; and the same remark must be true of these organs, when percussed within the body. A full sound, produced by percussion of the thorax or abdomen, indicates the existence of air beneath, through a space of at least several inches in extent, in every direction. A thoroughly empty sound, resembling the percussion-sound of the thigh, shews that there is neither air, nor any other gaseous body, beneath the percussed spot, but that solid fleshy bodies, or fluids, are present there.

The quantity of fluid, etc., necessary to make the percussion-sound, of any particular part of the thorax or abdomen, resemble the percussion-sound of the thigh, depends upon the elasticity of the walls of the

thorax and the abdomen, at the point percussed, and upon the condition of the parts in the space behind the fluid, etc. The more elastic the walls are, the more readily will vibrations spread through the subjacent fluid, etc., into any space containing air, behind or around the fluid, etc.; the greater this space is, the louder will the sound be.

The completely empty percussion-sound — the thigh-percussion-sound—heard at any yielding part of the walls of the thorax, or the abdomen, indicates that no air is present in that part, for a space several inches in depth, and one inch or more in circumference.

Of this fact we may obtain the proof, by placing a portion of lung, or intestine, which contains air, under water, and then percussing its surface by the aid of the pleximeter; it will be found that the lung, or the intestine, yields its proper sound, even though it be sunk several inches deep; but the nearer it is brought up to the surface, the more distinct the sound becomes.

Second Class:—*Percussion-sounds. Clear—Dull.* —The words clear and dull, or muffled, will be taken in their usual significations. The sound of a drum becomes duller, when covered by cloth; in the same way, we find the percussion-sound of the walls of the thorax and abdomen clear, in proportion as they are thin and elastic. When air is present, beneath a thin and elastic portion of the thoracic walls, through a space about an inch in breadth, and not more than a few lines deep, and the remainder of the thoracic cavity is filled with fluid, or con-

solidated lung-parenchyma, the percussion-sound over that portion will be perfectly clear, but very empty. On the other hand, any portion of the lung, situated immediately beneath the thoracic walls, which has a surface not less in circumference than a pleximeter, and half an inch of thickness, will yield a full but quickly muffled percussion-sound, if it be deprived of air, whilst the rest of the thorax is filled with the normally distended lung.

A small portion of intestine, lying against the abdominal walls, and filled with air (the air having being expelled from the remainder of the intestines by peritoneal effusion), yields a very clear but empty sound. A portion of intestine, containing air, which lies in part beneath the liver, and in part in contact with the abdominal walls, yields, when percussion is practised over the border of the liver, a muffled sound; but when the pleximeter is placed beneath the border of the liver, the sound becomes perfectly clear.

The truth of the above statements may be readily shown, by experiments on the dead body. A hepatized lung taken out of the body, yields the thigh-percussion-sound; but if only a small part of it contain air, and this part be percussed, it gives a clear, but very abrupt sound, having little resonance, and which, according to my views, must be called empty. Percussion of a superficially infiltrated portion of lung, if it be equal in size to a pleximeter, and the lung otherwise healthy, yields a duller sound than the rest of the surface: and the thicker the portion deprived of air, the duller is the sound. The hepa-

tized part of the lung may be as much as six inches thick, before the sound of the portion of lung beneath it, which contains air, is altogether obliterated; and before the sound becomes as dull as the thigh-percussion-sound. A portion of intestine containing air, and placed under water, so as to remain partially exposed to the air at the surface, yields, when percussed there, as clear a sound as though no part of it were covered by water; the sound of the intestine immersed in the water, and percussed through the water, is muffled, and becomes more so, in proportion as the intestine is sunk deeper.

From all this it is evident that the expressions, full and clear, dull and empty, have different significations. A percussion-sound may be full and clear, and also full and dull, empty and clear, and empty and dull. A completely dull, and a completely empty sound, have naturally the same significance, and they are represented by the thigh-percussion-sound. As a sound becomes duller, it at the same time always becomes emptier. A less full sound, however, is not necessarily a dull sound; a sound may be very empty, and yet perfectly clear.

The degree of dulness of the percussion-sound, does not always enable us to judge accurately of the thickness of the non-resonant parts, beneath the spot percussed; for the dulness depends in part also, upon the thickness and elasticity of the parts percussed, and upon the condition of the space containing air, behind the non-resonant parts.

Third Class:—*Percussion-sounds. Tympanitic — Non-tympanitic.* The tympanitic percussion-

sound, passes gradually into the non-tympanitic, just as the full into the empty, and the clear into the dull; no distinct line of demarcation can be drawn between them.

The non-tympanitic is represented by the sound which percussion produces at those parts of the thorax, beneath which lies healthy lung, normally distended by air. An abnormally distended lung, as in vesicular emphysema, gives us at one time a tympanitic, at another, a non-tympanitic sound. A partial emphysema in the midst of lung deprived of air (as happens in pneumonia, where not unfrequently the tissue around the hepatized portion, and especially at the borders of the lung, is emphysematous) generally produces a tympanitic-sound; but if the whole of the lung is emphysematous, the sound is seldom distinctly tympanitic. If the lung contain less than its normal quantity of air, it yields a sound which approaches to the tympanitic, or is distinctly tympanitic. The sound is, moreover, in many cases remarkably tympanitic, even when the diminution of the quantity of air in the lung is the effect of an increase in its fluid or solid constituents; and this, too, whether the lung retains its normal volume, or becomes larger than natural. When the lung is much reduced in volume by compression, but still contains air, its sound is invariably tympanitic.

That the lungs, partially deprived of air, should yield a tympanitic, and, when the quantity of air in them is increased, a non-tympanitic sound, appears opposed to the laws of physics. The fact however is certain, and is corroborated both by experiments on the

*dead body (which will be presently referred to), and also by this constant phenomenon, viz.: that when the lower portion of a lung is entirely compressed by any pleuritic effusion, and its upper portion reduced in volume, the percussion-sound at the upper part of the thorax is distinctly tympanitic.**

When the walls of the thorax are thin and yielding, the percussion-sound may remain tympanitic, even though the quantity of air in the lung be very small; this fact we occasionally observe in cases of pneumonia and tubercular infiltration. The condensed portions of lung, beneath the thoracic walls thus thin and yielding, give, in some cases, a distinctly tympanitic, though very empty, and not very loud sound. The percussion-sound is seldom tympanitic, when the walls of the thorax are dense and unyielding.

When we percuss a lung—which contains a greater

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* Dr. C. J. B. Williams (Lectures on Diseases of the Chest) gives a different explanation of the tympanitic sound thus produced. He says: We shall understand how this sound arises, by observing the tracheal-sound, which is produced by percussing the trachea with the finger above the sternum. The trachea passes behind the sternum, and there divides into two large branches, which lie about one or two inches beneath the clavicles; they are here covered by porous lung, and percussion over them consequently excites no resonance. But if this portion of the lung should either be condensed by effusion, or hepatized, then percussion at once gives us a hollow tubular sound. The reason why we do not more frequently meet with the phenomenon is, that the compression, or consolidation of the upper lobe of the lung, is seldom complete enough to produce it.—Direct experiments prove that this explanation is incorrect.

quantity of air in some parts of its structure than in others, that is, in which groups of strongly distended air-cells are mingled with others that are less distended, or contain scarcely any air—we find that it yields a sound in part tympanitic, and in part non-tympanitic.

Cavities of the lungs that contain air, lie near the surface, and are about equal to the size of a pleximeter, invariably yield a tympanitic sound, when surrounded by consolidated lung tissue; but when they are surrounded by healthy lung-tissue, the sound is less tympanitic, or even non-tympanitic.

In pneumothorax, the walls of the thorax, if they are not much distended, yield a tympanitic-sound; but if much distended, their sound is almost constantly non-tympanitic.

When the intestines contain gas, but are not forcibly distended by it, nor compressed by the abdominal walls, they always render the percussion-sound of the abdomen tympanitic; but if they are much distended, or compressed by the muscles of the abdomen, the sound becomes less, or even non-tympanitic.

What has been here said respecting the causes which render percussion-sound tympanitic or non-tympanitic, may be proved by experiments. A healthy lung taken out of the body, and fully distended with air, when percussed through a pleximeter, yields a clear, full, non-tympanitic sound; but when not inflated, though it contain little air, and be even somewhat collapsed, it gives a clear, full, and tolerably distinct tympanitic sound. The percussion-sound is tympanitic also when water, even to a considerable

amount, has been injected, but not too forcibly, into a collapsed or inflated lung; if the quantity of water, however, be much increased, the sound becomes emptier and less clear. An emphysematous lung, which remains distended when taken out of the body, but is not otherwise changed in structure, has the same sound as a healthy inflated lung. Interlobular emphysema gives a decidedly non-tympanitic, and less clear sound, than a healthy inflated lung.

The sound of any part of a lung, which is infiltrated with serum, blood, or tubercular matter, but not entirely deprived of air, is tympanitic, and more or less empty and dull, according to the quantity of air present in it. The sound of a lung, containing merely a few solitary tubercles, does not differ from that of a healthy lung.

An inflated lung percussed through the medium of a piece of liver, sounds non-tympanitic, a collapsed lung containing air, tympanitic; but in both cases, the dull and empty character of the sound is proportionate to the thickness of the liver employed; and this must be very thick before the tympanitic sound disappears. The same results ensue, when a piece of hepatized lung is used instead of liver, or when the lung is placed under water, and percussion made on the surface of the water.

A healthy lung strongly inflated within the thorax, so as to be made to press against its walls, gives a full, clear, but non-tympanitic sound, at every part where it comes in contact with the walls. In performing this experiment, it is necessary to make one or more openings into the thorax, in order to ensure

the inflation of the lung and its contact with the walls, by letting out any gases which may have been accidentally evolved after death.

If water be forced through the trachea into a lung, inflated in the manner described above, or after the lung has again collapsed and a portion of its air escaped—so as to cause a kind of artificial œdema of the lungs—it will be found, that the sound remains much the same as in a lung containing no water; and that a very considerable quantity of water must be thrown into the lung before any dulness can be detected in its percussion-sound. Whatever be the amount of the water injected, the sound never becomes completely dull.

The percussion-sound invariably becomes full and clear, and at the same time either slightly or markedly tympanitic, whenever air is forced into the pleural cavity, so as to compress the lung. If water be thrown into the pleura, the percussion-sound is clear, and either approaches the tympanitic, or is distinctly tympanitic, at every point at which the lung touches the walls of the thorax: where the water comes in contact with the walls, the sound is dull, and in proportion to the amount of water present. If the quantity of water be not very considerable, the sound is often tympanitic.

Strong inflation of the stomach, or of a portion of intestine, causes these organs, when percussed, to yield a dull sound, approaching the non-tympanitic; but when gently inflated, they give a clear tympanitic sound, if care be taken not to press the pleximeter so firmly as to distend their coats. A stomach, or

portion of intestine, filled in part with air and in part with water, yields the same sound as it would do if entirely filled with air; but here, also, a clear tympanitic sound will not be produced, if the coats have been rendered tense.

When we percuss an intestine, through the medium of non-resonant organic bodies, as through portions of liver or spleen, or through water, we find the sound behaves itself exactly as in the experiment referred to with the lung. Percussion of an intestine, through the medium of a healthy portion of lung, produces a modified sound, composed of the sound of the lung, and of the sound of the stomach, and generally of a tympanitic character.

A dull sound, either slightly or not at all tympanitic, is produced by percussion of the abdominal walls, when they are stiffened after death, and firmly compress the intestines, even though these last contain a considerable quantity of gas, and gave a distinctly tympanitic sound before death, *i.e.*, when they were not so firmly compressed. If the abdominal walls be lax after death, then the sound is tympanitic, and it remains so although a considerable amount of fluid be present in the peritoneal cavity; the same thing may be observed when percussion is made over the liver, a portion of intestine which contains air, lying behind it.

It is thus proved, both by observations on the living body and by researches in the dead, that the percussion-sound is invariably tympanitic, when the parietes of the organ, which contains the air, are not stretched; but that, on the contrary, when they are firmly

stretched, the percussion-sound becomes less, or not at all tympanitic, and even dull.

Thus the fully distended stomach, the strongly inflated lungs, the tense thorax (as in pneumothorax), the firmly contracted abdominal walls, produce a non-tympanitic or merely an indistinctly tympanitic sound; whilst, on the other hand, the relaxed stomach, the collapsed lungs, the compressible abdominal walls, give a distinctly tympanitic sound. Respecting the cause of these remarkable facts, it may be observed, that the tympanitic percussion-sound approaches in character to a tone (*Klang*), the non-tympanitic, to a murmur (*Geräusch*). A greater homogeneity of vibrations appears necessary for the production of a tympanitic, than of a non-tympanitic sound. When percussion is made upon a non-distended stomach, it is the air alone within it which produces the sound; but if the stomach be strongly distended, its coats also vibrate, and these vibrations seem to interfere with those of the contained air, and thus to be the cause of the dull non-tympanitic sound.*

Fourth Class:—*Percussion Sounds,—High—Low.*—Variations in pitch, are most readily detected

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* Dr. Kürschner (Schmidt's *Jahrbücher*, 32 B., 1 H.) explains the cause of the tympanitic, and the non-tympanitic sounds, by an observation of Savart's. Savart found that the more a membrane was stretched, the less readily could vibrations be excited in it. The stomach containing air sounds tympanitic, when its coats are so slack that no sonorous vibrations can be excited in them.—I still believe that my explanation is correct.

in sounds which are clear; but they are of little value in practice. This fact may be readily shewn by experiments. A narrow intestine gives a deeper sound than a wide one, but its pitch varies with every change in the condition of the intestine. The same fact is observed in percussion of the lungs. It is worthy of remark, however, that a change in the pitch of the sound—generally an elevation of it—often precedes the conversion of a non-tympanitic lung-sound, into a tympanitic; this sign may be of value, if there be no other difference observable in the percussion-sound. In fact, it occasionally happens, that the presence of tubercles, in the upper part of a lung, may be diagnosed by the different pitch of the percussion-sound, over the corresponding part of the other lung.

The Metallic Ringing Percussion-sound, and the Cracked-pot-sound.—These sounds cannot be classed in any of the foregoing divisions.

Piorry calls the metallic ringing percussion-sound, "water-sound"—*son humorique, hydropneumatique*, —from a belief that the presence of air and water together, is necessary to produce it. It is the sound which we elicit, by striking empty, or nearly empty vessels. That the presence of water is not required for its production, may be demonstrated by percussing (either with or without a pleximeter) a stomach filled with air, and not containing one drop of water: this experiment succeeds best, when the coats of the stomach are not made too tense. But the sound is also heard, when the stomach contains both air and water, and may be also produced in

wide, and even narrow portions of intestine. It may be frequently observed over large thoracic cavities which contain air, and also when air, or other gases, are present in the pleural cavity.

The cracked-pot-sound may be closely imitated, by percussing a portion of intestine inflated with air, pressing it at the same time with the pleximeter, so as to make its anterior wall approach the posterior; and also, by bringing the palms of the hands together, and then striking the back of one of them against the knee.

The cracked-pot-sound is heard in the thorax, over tolerably large and superficially situated cavities, which contain air, and communicate with the bronchial tubes. When the percussion is forcible, or the thoracic walls flexible, the cavity is compressed at each stroke, and a portion of air suddenly driven out of it into the bronchial tubes; the hissing murmur, caused by the escaping air, is mixed up with the ordinary percussion-sound of cavities, and this compound represents the cracked-pot-sound. The air driven out, sometimes passes through fluids, or the fluid in the cavity is disturbed by the percussion-stroke, and a sound, similar to the movement of saliva in the mouth, is then produced.

It is said, that the cracked-pot-sound may be heard in children, in whose lungs there are no cavities. I have never observed it there myself. It cannot be produced, unless the person percussed, keeps his mouth open; it is not heard, if the mouth, and more particularly if, with the mouth, the nostrils also be closed; closure of the mouth and nostrils

must have a tendency to prevent that slight depression, which is caused by each percussion-stroke.

Besides these sounds, Piorry describes another—the *hydatid-sound.* This is, in fact, not a sound; it arises from certain vibrations, the presence of which may be ascertained by the hand, or the points of the fingers.* An excellent notion of Piorry's hydatid-sound, may be obtained by percussing a stomach completely filled with water, and held free in the air. Tapping a repeater, held in the hand, also produces it, that is to say, the vibrations of its main spring are thereby rendered sensible. Piorry and Briançon assert, that these peculiar vibrations are only to be observed over hydatid cysts, and depend upon the tremulous movements of the hydatids. I do not know if any one else has made similar observations. The experiment with the stomach shows that the presence of hydatids is not necessary for the production of the sound. It may also almost invariably be observed, in cases of peritoneal effusions, when the abdomen is tense, and its walls not very thick. The conditions necessary for the production of this sound, are not so often met with in ovarian dropsy, as in ascites; an hydatid cyst, giving rise to it, must be looked upon as a phenomenon of very rare occurrence.

B. THE RESISTANCE FELT IN PERCUSSION.

Piorry was the first observer who pointed out the

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* A dull sound is heard through the stethoscope.

fact, that in percussing the various organs, different degrees of resistance are felt by the fingers; and it would seem as though he considered this resistance of more importance than the percussion-sound.

The various degrees of resistance, offered by the different organs when percussed, may be learned on the dead body. An healthy, air-containing lung, offers no resistance when the pleximeter is so held as merely to touch, without pressing upon its surface; but if the lung has been rendered dense and firm, through infiltrations of serum, blood, or tubercular matter, the resistance becomes perceptible, and is greater in proportion as these matters are more abundant, and the quantity of air in the lung diminished. A hardened lung offers a greater resistance than a soft lung.

Resistance is not felt in percussion of the stomach and intestines, unless the coats of these organs are tense; the resistance increases with the tension.

The resistance of organs not containing air, is regulated by the degree of their firmness; that of the thoracic walls, is greater, *per se*, in proportion to the thickness and unyielding nature of the ribs, and the narrowness of the intercostal spaces. The resistance of the abdominal walls, is increased by their tension and firmness. The thicker and firmer the ribs, the narrower the intercostal spaces, and the tenser the abdominal walls, the less will be the difference, between the degree of resistance felt over the organs of the thorax and abdomen, in their normal condition, and the resistance which they offer in their abnormal conditions.

The healthy lung offers no resistance; the resistance felt at all parts of the thorax, wherever healthy lung comes in contact with its walls, is caused by the walls themselves. Air present in cavities, or in the pleura, likewise offers no resistance, except in those cases in which it causes distension of the thoracic walls.

When the intercostal spaces are enlarged, as in pneumothorax, or in emphysema of the lungs, we find that the thoracic walls are sensibly depressed at each percussion-stroke, but quickly regain their previous form, the thorax being more elastic than usual. The same thing is observed, even in a healthy condition of the lungs, when the ribs are thin and the intercostal spaces large; only, in this case, the resistance is less than in pneumothorax and emphysema of the lungs. Should it happen in pneumothorax, or, what is rarer, in pulmonary emphysema, that the intercostal spaces are not enlarged, or that, notwithstanding such enlargement, the ribs themselves are inflexible, then no sensation of any yielding can be felt.

The lungs become resistant to percussion, when infiltrated with blood, serum, tubercular matter, etc.; but it is impossible to define accurately what degree of distension and consistence of the infiltrated lung is necessary to render this resistance perceptible through the thoracic walls, as so much depends upon their flexibility. When an entire lung, or a considerable portion of it, is hepatized, or infiltrated with tubercle, from before backwards, and solidified, the resistance felt at the corresponding parts of the

thoracic walls is as great, or even greater, than that felt over the hepatic region, in ordinary enlargement of the liver.

The resistance offered by the thorax is greatest when its walls are rendered tense, and its intercostal spaces distended by pleuritic effusions; little resistance is caused by effusions which do not make tense the walls which contain them.

The resistance of the heart, the liver, and the spleen, increases in proportion to their solidity, and to the force with which they are pressed against the walls of the thorax.

The difference of resistance felt in percussion, enables us to decide whether an abdominal swelling is caused by the presence of air in the intestines, or of fluid in the peritoneum. Encysted fluids, which render the walls of the cyst which contains them tense, offer, through the abdominal walls, the same resistance as tolerably firm fleshy bodies.

CHAP. II.

AUSCULTATION.

The sounds produced in the thorax by the movement of the organs within it, are seldom so loud as to be perceptible, until the ear is placed in contact with its walls. Those very rare cases in which the sounds are audible at a distance from the thorax, do not seem to have attracted the attention of early observers; and this is not surprising, so long as physicians, in their interpretation of the phenomena of diseases, did not proceed by special investigation of the conditions of the different organs of the human body.

The want of some new method of investigating the abnormal conditions of the internal organs, was made manifest by the progress of pathological anatomy; this was the reason, that rendered Auenbrugger's "Inventum Novum" so welcome to Corvisart, who first brought it into general notice; previously to him, Auenbrugger's discovery had been thought of little value, and was, indeed, almost forgotten. Corvisart was well acquainted with the morbid anatomy of the heart, and of the organs of respiration, but he was ignorant of the signs by which to discriminate the different diseased conditions of these hidden organs. Auenbrugger's discovery, therefore,

which so essentially aided the diagnosis of diseases of the thoracic organs, could not be otherwise than most welcome to him.

Corvisart was accustomed, in cases where the movements of the heart could not be satisfactorily ascertained by the hand, to place his ear over the cardiac region; and thus he practised immediate auscultation. His pupils followed his example; but this new method of diagnosing the heart's movements does not appear, for a long period, to have been of much general benefit, on account probably of its being but little practised. The immense importance of auscultation was at length demonstrated to the world by Laennec, who, after spending three years in the investigation of its phenomena, published those results which have made his name immortal. His labours gave a new direction and a new impulse to the spirit of investigation in the physicians of France, and afterwards of most other countries. His observations and theories have been, and will be again and again, subjected to criticism; and it becomes the duty of every physician who has the opportunity, to distinguish in them what is certain from what is doubtful or incorrect.

Laennec, at first, considered auscultation by the stethoscope as a method of investigation totally distinct from immediate auscultation; but he appears to have afterwards modified this opinion, still, however, believing that he who did not use the stethoscope, but trusted to the unaided ear, could never attain to any certainty in diagnosis. Notwithstanding this, immediate auscultation has been practised by many phy-

sicians, and has even been thought to have advantages over mediate auscultation.

I do not think it is necessary to repeat all that has been said, from time to time, respecting the advantages and disadvantages of these two methods of auscultation. Sounds are heard louder by the unaided ear, than through the stethoscope; but the ear cannot be applied to every part of the thorax, and the disease may be of such a character, or the person to be examined in such a condition, as to render immediate auscultation very repulsive to the physician.

My own opinion is, that the stethoscope is indispensable, and that every physician must make himself acquainted with its use; but he must, nevertheless, also study immediate auscultation, for it frequently happens, from the situation of the patient, or the position of his bed, that the ear can be more readily applied to the chest than the stethoscope. Physicians, therefore, must practise both mediate and immediate auscultation.

I do not myself agree with Laennec, that the auscultatory sounds, which have reference to the voice, are rendered more distinct by the stethoscope than they are without it; nor do I find that patients dread the use of the stethoscope more than they do the application of the ear to the thorax; or that this last procedure is more agreeable to them than the employment of the stethoscope. When the patient lies in bed, the physician will, with very few exceptions, find it most convenient to use the stethoscope: but if the patient sits or stands, immediate auscultation, particularly of the back, is very easily performed.

The form of the stethoscope, and the nature of the wood it is made of, are often objects of much attention with those who are not well acquainted with auscultation; but in regard to conducting power at least, the choice of the wood is quite indifferent, for the greater part of the sound traverses the air in the stethoscope, and not the wood. The lighter the wood, the more convenient is the instrument, both for the physician and the patient. Whether it be long or short, formed of one or two pieces, screwed or slipped together, is equally matter of indifference as respects hearing. The funnel-shaped end should not have too large a diameter, for in such case, it cannot be accurately applied to the chest; and besides, too great hollowness may produce modifications in certain sounds. It is sufficient, if the funnel has a diameter of about one inch. The ear-piece may be convex, concave, or plane, provided the disk forming it be large enough to close the ear completely. The shortness of Piorry's instrument often renders its application difficult, and at times impossible; and this is found to be particularly the case in patients whose movements are not free; on this account, I use a stethoscope about a foot long.

Whether the auscultation be mediate or immediate, every care should be taken not to render it wearisome to the patient; and no more pressure should be made by the ear, or by the stethoscope, than is necessary to exclude all communication with the external air.

Beginners, especially, should be cautioned not to press heavily; while listening to the sound, they

are apt to forget their position, and to allow the whole weight of the head to fall upon the chest of the patient, which would be sufficient to impede respiration, even in the healthy, and much more so in those suffering from disease; the stethoscope, moreover, is liable to produce pain, in consequence of the small extent of surface which receives its pressure. By paying attention to his position, in using the stethoscope, the auscultator will cause little annoyance, even to the most sensitive individual, provided the latter be neither prejudiced against, nor dreads its use.

In order to derive all the information from auscultation which it can afford us, we must not be contented with the examination of one, or even of several, parts of the thorax; every part must be examined, and a comparison then made of the results obtained.

CHAP. III.

THE AUSCULTATORY PHENOMENA OF THE ORGANS OF RESPIRATION.

THE phenomena observable in the respiratory organs by auscultation, are: the thoracic voice; the murmurs caused by the passing to and fro of the air during inspiration and expiration; and the murmur produced by the rubbing of roughened pleural surfaces.

I. AUSCULTATION OF THE VOICE.

The observer soon learns by experience, that the thoracic voice is heard very differently in different individuals: in one it will be found strong and clear; and in another, who speaks equally loud, it will be merely represented by an indistinct humming (*Summen*), or perhaps no trace whatever of the voice will be audible. The voice of the same individual, whether his thoracic organs be healthy or unhealthy, is not heard equally loud at all parts of the thorax.

Auscultation teaches us that the voice may be heard in the thorax, in the most varying degrees of strength and clearness, and even up to a point where it seems to pass directly into the ear of the auscultator; numerous other variations in the timbre, etc., of the voice are also to be remarked.

The Strength and Clearness of the Thoracic Voice.—It is necessary to make a distinction between strength and clearness of the voice. The voice may be distinctly heard in the thorax, without being strong; and, on the other hand, a strong voice is not necessarily a clear one.

There are several degrees, both in strength and clearness of the thoracic voice; the voice, as heard over the larynx, may be taken as a standard by which to measure these degrees. Generally, the voice over the thorax is less strong and clear than it is over the larynx, it is seldom equally strong and clear, and very rarely is it stronger and clearer. I divide the thoracic voice, in respect to strength, into weak and strong; and I call it strong, when it not only emits sound, but also produces a degree of concussion in the ear. If this concussion does not strike deep into the ear. I term the voice weak.*

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* The vibrations felt by the hand, when laid upon the thorax of a person speaking, have not the same significance, as the vibrations communicated to the ear, in auscultation of the thoracic voice. In persons whose voices are deep, the vibrations may be often felt very strong, and yet no voice, but merely an indistinct humming, is appreciable by auscultation, and no vibrations pass into the ear. On the other hand, in persons whose voices are high, scarcely any vibration of the thorax can be felt by the hand, although, by auscultation, the thoracic voice may be clearly heard, and penetrate far into the ear.

These phenomena are in unison with the known facts, that the sonorous vibrations of solid bodies are more readily sensible to the touch, the lower the tones are which produce them; and that vibrations may be felt by the hand, when they are not rapid enough to communicate sound to the ear. The vibrations of

We have no such standard by which to mark the clearness and the dulness of the thoracic voice. The voice, however, may be always called clear when its articulation is audible. Yet the voice may be very clear, though the articulation be not particularly distinct.

In the normal state of the respiratory organs of many individuals, we hear no thoracic voice, but merely a humming; this humming is loudest between the scapulæ and the vertebral column, less loud beneath the clavicles, and it becomes gradually lost as we pass down towards the lower parts of the thorax. When the voice is deep, and the respiratory organs healthy, it is frequently heard strong, and tolerably clear, between the upper half of the scapulæ and the vertebral column, but is rarely attended with any distinct articulation. When the voice is acute, it is frequently also weak and clear.

The voice, again, often appears less strong and clear beneath the clavicles (the respiratory organs being healthy), than between the scapulæ and vertebral column; and still weaker and duller in the axillæ. In other parts of the thorax, no voice, but merely a humming, is heard, and sometimes not even a humming.

When the respiratory organs are diseased, there

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the thoracic walls are communicated to the ear, and may be slow or rapid; but if they are not appreciable as a tone, or form only a very weak tone, no sensation of vibration will pass deep into the ear; a sense of concussion only being felt in the external ear, or in the head.

are no parts of the thorax, where the voice may not be heard strong and clear; generally speaking, it is so heard at those parts where cavities, pneumonic or tubercular infiltrations, or pleuritic effusions exist; but this does not happen in every case, nor during the whole period of the existence of such diseased conditions of the organs.

Variations in the Strength and Clearness of the Thoracic Voice cannot be explained by the Laws of Conduction of Sound.—Laennec attributed the variations observed in the strength and clearness of the thoracic voice to changes in the sound-conducting power of the lung-parenchyma; he considered the lung, in its normal state, to be a bad conductor of sound, but that its conducting power was increased by its consolidation and infiltration, or by the presence of fluids in the pleura—an explanation quite in accordance with the generally received opinion, that solid bodies conduct sound better than air.

This opinion has prevailed in France up to the present time.

Now, if we repeatedly auscultate the thorax of a person suffering from hepatization of a lung, we shall find that the thoracic voice is at one time increased, and at another diminished in force, without any alteration—discoverable by percussion or other means—having taken place in the condition of the hepatized part. This alternate presence and absence of the thoracic voice at the same part of the thorax, when the lung is hepatized, is a well-known and common occurrence. Everyone who has had much experience in the auscultation of pneumonia, must

have observed the increased voice, bronchophony, to appear and disappear several times in the course of a few minutes.

The phenomenon is opposed to the idea of the bronchophony depending upon a superior conducting power of sound, inherent in hepatized lung; and whoever maintains the correctness of such an idea, is bound to explain this anomaly.

The voice, as we all know, though it may have disappeared, will return after a deep-drawn breath, and still more readily if the patient coughs; and it will again disappear if he remain tranquil for a short time, without coughing or expectorating. The conclusion to be drawn from this is, that the voice is heard through the hepatized parts, when the bronchial tubes passing into them are not obliterated by fluids, but contain air; and that, on the other hand, it disappears when the tubes are blocked up by mucus, etc. This explanation of the absence and presence of the thoracic voice, in no way removes the difficulty as to the increased sound-conducting power of the hepatized lung; if this power were really increased, it would become a matter of indifference whether the bronchial tubes contained air or fluids.

It also appears doubtful—as in the case of hepatization of the lung—whether an increase in the sound-conducting power of the lung takes place in the course of pleuritic effusions; for the voice becomes weaker in proportion as the effusion progresses, the reverse of which ought to happen, if the effusion were a better conductor of sound.

The following remarks on the sound-conducting

power of bodies and its conditions, do not justify the supposition that bronchophony depends upon an increased sound-conducting power of the hepatized lung and of fluids. The human voice, and every other sound which is formed or propagated in the air, is heard furthest in the air. A sound excited in the air, is heard very indistinctly, or not at all, by a person under water; and a sound in one room passes with difficulty into another, being interrupted by the walls. Anyone wishing to weaken his hearing, stops his ears.

On the other hand, the slightest scratching at one end of a long rod, may be heard, if the ear be brought in contact with the other; while no sound whatever is audible in the air, although the ear be brought much nearer to that end of the rod whence the sound proceeds. The sound caused by striking two stones together, under water, is distinctly heard there, and even causes a disagreeable sensation; whilst, out of the water, it can be scarcely recognized.

These facts show that sound does not pass readily from dense bodies into the air, or from the air into dense bodies. Physics, also, teach us that sound is always reflected, in passing from one medium into another, and that less sound enters into the new medium than would have been propagated through a corresponding space of the one in which it was originally excited; the more dissimilar the media are, in respect of density and cohesion, the greater is the reflection of the sound, and the less freely does it pass from the one into the other.

The ticking of a watch is heard to a greater dis-

tance through a rod, than through the air, because no part of the sound passes off from the rod into the surrounding air, but remains wholly concentrated in it; the sound, on the contrary, which passes immediately from the watch into the air, spreads out in every direction, and thus impinges upon a greater extent of matter. The experiment with the rod, however, does not prove that wood is a better conductor of sound than air. The difference in the conducting power of air, wood, and other bodies, has not been experimentally determined. Researches made for this purpose, must be of such a nature as to show the results of one and the same sound in two or more media, which have like form and volume, and are placed in a similar relation to the parts surrounding them; the distances at which the sounds are heard through each medium, and their force, must also be compared.

Let any one, for example, so place the end of a wooden tube on a watch, that the whole rim may be in contact with it, and then listen at the other end; he will hear the ticking at the same moment through the wood, and through the air in the tube. If a solid cylinder of wood be now fitted into the tube, and it be placed as before, the ticking will be heard passing through the wood of the tube, and through that of the solid cylinder. Now, if wood were a better conductor of sound than air, the ticking ought to be heard more clearly in the latter than in the former case; but any one may readily convince himself that the reverse of this is the fact.

It is a remarkable circumstance, that auscultators

should make use of a hollow tube, and not of a solid cylinder, and yet assert that dense bodies are better conductors of sound than air.

There is no doubt that the voice passes into the parenchyma of the lungs through the medium of the air contained in the trachea and bronchial tubes; for if it were propagated along the walls of the trachea, it would spread equally well through the general coverings over the thorax. When the lungs are healthy, and the air passes uninterruptedly into the air-cells, the voice reaches further than when the lung is hepatized, or compressed by fluids, that is, than when the air-cells and finer bronchial tubes contain no air. The more solid a body is, the more difficult is the passage of sound from the air into it; and hence sound passes more readily from the air of the air-cells and bronchial tubes into the parenchyma of a healthy lung, than it does from the air of the larger bronchial tubes into the consolidated tissue of a hepatized lung.

The conducting power of healthy and of hepatized lung, and of fluids, may be readily shown in the following manner. Let one person direct his voice into a stethoscope placed upon a healthy lung, removed from the body, while another auscultates with a second stethoscope; by gradually placing the stethoscopes at different distances, the exact distance through which the voice can be heard in the lung, will be at last ascertained; similar experiments may be afterwards performed with hepatized lung, and lung compressed by fluids.

Repeated experiments of this nature, have in-

variably demonstrated to me that sound is heard somewhat further through healthy than through hepatized lung. The difference in this respect is remarkable.

In accordance with the above facts, I have come to the conclusion that: Variations in the strength and clearness of the thoracic voice cannot be explained by differences in the sound-conducting power of normal and abnormal lung parenchyma.

The Variations in Strength and Clearness of the Thoracic Voice explained by the Laws of Consonance. —If a sound is heard as distinctly at a distance from, as at the spot where it originates, one of these two things must have happened: either its diffusion has been prevented, and it has remained concentrated in the passage, or it has been reproduced by consonance, and thus increased in strength; and if the sound be heard louder at a distance from, than at its origin, it must also have gained increase by consonance.

Consonance is a well known phenomenon. A guitar-string yields a musical note, when a similar note is sounded upon another instrument in its neighbourhood, or even by the human voice. A tuning-fork held in the air, sounds much more feebly than when laid on a table; the table strengthens the tone, and yields similar vibrations, and thus consonates with the tuning-fork.

The sound of a jew's-harp is scarcely heard in the open air, but becomes distinctly audible when made to vibrate within the mouth—its sound is strengthened, in consequence of the air in the mouth consonating with its vibrations.

When, as occasionally happens, the voice is heard louder at some part of the thorax than over the larynx, it must have derived its increased strength from consonance within the thorax. Variations in the strength and clearness of the thoracic voice may thus be explained by changes in the force of its consonance within the thorax; and it therefore becomes necessary for us to inquire here, what parts within the thorax consonate with the voice, and what circumstances cause variations in the consonance.

The voice, as it proceeds from the mouth, is formed of the original sounds of the larynx, and of the consonant sounds of the throat and of the cavities of the mouth and nose. This we learn from the changes which the voice undergoes by opening and closing the mouth or the nose, while the condition of the larynx remains unaltered. It is well known, that the pitch of the voice is determined by the larynx, and that it is not affected by the opening or closing of the mouth or nose; but the articulation of the voice takes place in the mouth, and certain modifications in its timbre depend upon the form and size of the mouth and nasal cavities, and particularly upon the circumstance of their being opened or closed.

Now, since it is evident that the air in the throat, the mouth, and the nasal cavities, consonates with the sound formed in the larynx, we cannot doubt that the air in the trachea, the bronchial tubes, etc., may likewise become consonant with sounds originating in the larynx. The air in the thorax, and not the parenchyma of the lungs, is the consonating body:

the parenchyma is ill-adapted for consonance, being neither firm nor tense in structure.

Those bodies in which musical vibrations are most readily excited, such as the air, musical chords, membranes, bars, plates, etc., also consonate most readily.

Air only consonates when confined in a given space. The human voice, and every other sound, is much weaker in the open air than in a room. The air contained within the sounding-board of a guitar, a violin, or a clavier, consonates with the tones produced by their strings, but the free air around does not strengthen their tones.

The force of the consonance depends upon the form and size of the enclosed space, and upon the nature of the walls forming it; the consonance, for example, is stronger, the more completely the sound is reflected by the walls; hence a space bounded by solid walls yields the loudest consonance, whilst those formed of linen, as in a tent, add little to the force of the sound. The cause of the increase of sound in a speaking-trumpet is well known.

The air enclosed in a given space does not consonate with every sound; and although several sounds or murmurs may consonate therein, it will be found that they do not do so with equal force and clearness. Consonating bodies only respond to those tones which they themselves are able to produce, or to vibrations forming some aliquot part of such tones.*

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* Die Naturlehre, nach ihrem gegenwärtigen Zustande, von Baumgartner und Ettingshausen.

The following conclusions respecting the consonance of the thoracic voice, may be drawn from a consideration of these physical data: the air contained in the trachea and bronchial tubes, consonates with the voice, in so far as the walls confining it have, in respect of their power of reflecting sound, a similar or analogous condition to the walls of the larynx, of the mouth, and of the nasal cavities. In the trachea, the walls of which are formed of cartilaginous rings, the consonance of the voice is nearly as forcible as the voice itself, heard in the larynx; and it can be little less so in the right and left bronchi.

As the bronchial tubes pass into the parenchyma of the lungs, their cartilaginous rings gradually disappear, the cartilaginous structure existing at last only as irregular thin plates, lying in a fibrous tissue; the finer divisions of the bronchial tubes are merely thin membranous canals. The consonance of the voice is consequently much feebler in the bronchial tubes, which run into the parenchyma of the lungs, than in the trachea, and, in fact, becomes weaker in proportion as the amount of cartilaginous structure diminishes.

The conditions requisite for the production of increased consonance of the voice in the bronchial tubes, which run into the parenchyma of the lungs, are these: either, the walls of the tubes must be cartilaginous, or, if membranous, the membrane must be very dense, or the tissue of the lung around the tubes must be deprived of air; in any of these cases, the walls of the bronchial tubes will reflect sound

more strongly, than in their normal condition. Of course the communication between the air in the tubes, and the air in the larynx, must remain uninterrupted.

It frequently happens, when musical vibrations—original or communicated—are excited in air in a confined space, that the walls themselves which surround the air, also vibrate in unison, and the more readily, the less rigid and unyielding they are. An organ-pipe vibrates, when the air within it sounds, and so does a speaking-trumpet. The trachea vibrates with every sound, and its vibrations are perceptible, even through several inches of firm fleshy layers. The walls of the bronchial tubes, running into the parenchyma of the lungs, likewise vibrate, when the voice consonates in them, just as the walls of the larynx do; the vibrations thus excited, may extend to the walls of the thorax, and even pass through several inches of thick fleshy parts, or of fluid, and be heard at the thorax as consonating sounds of the bronchial tubes.

Diseased Conditions of the Respiratory Organs, which, in accordance with the preceding explanations, will produce an increase in Strength or Clearness of the Thoracic Voice.

Among these may be classed:

1. All those diseases by which the parenchyma of the lungs is deprived of air, and rendered firm, dense, and solid. The walls of a bronchial tube, surrounded by such an abnormal parenchyma, reflect sound as well, or even better, than the wall of the trachea. And the reflection of the sound, and the force of its

consonance, is greater or less, in proportion to the density of the parenchyma.

The diseases which render the lung-parenchyma solid, are: pneumonia, tubercular infiltration, or pulmonary apoplexy—apoplexia pulmonum. In these diseases, no increase of the thoracic voice will occur, unless the air has been wholly expelled—or apparently so—from the air-cells, by the infiltrated matters: and the solidified portion of lung be of such a size as to contain at least one of the larger bronchial tubes, having air in it, and communicating freely with the larynx. The more extensively the lung is solidified, the more marked is the increase of the strength of the thoracic voice.

Pneumonia in its first stage, inflammation confined to a few lobules of the lung,—lobular hepatization,—œdema of the lungs, or limited effusion of blood into the lung-parenchyma, produce very slight, or no increase whatever, in the strength of the thoracic voice; neither do solitary tubercles, however numerous, provided the intervening parenchyma remains pervious to air. Effusion of blood into the lung-parenchyma—Laennec's pulmonary apoplexy—being of rare occurrence, and generally very limited in extent, seldom gives rise to increase of the thoracic voice. Its increase is very frequently observed in the course of extensive hepatizations and tubercular infiltrations of the lungs. It is also produced by the condensation of the lung, which remains after an unresolved hepatization, just as in hepatization itself. I have never found the lung substance completely deprived of air, in œdema of the lungs, unless the

lung was, at the same time, subjected to external pressure.*

II. ABNORMAL CONDITION OF THE LUNGS, IN WHICH THE PARENCHYMA IS DEPRIVED OF AIR BY COMPRESSION.

The walls of a bronchial tube surrounded by parenchyma, which is compressed and deprived of air, reflect sound as forcibly as the soft parts of the mouth; but compression of the lung will not give rise to the thoracic voice, unless the compressed portion be large enough to contain a bronchial tube, which, from its size and cartilaginous character, cannot undergo complete compression; the simply membranous bronchial tubes, may be entirely obliterated.

The lung-substance can be compressed by fluid or solid exudations, or by gas present in the pleura, by tumours, by enlargement of the heart, by pericardial effusions, aortic aneurisms, enlargement of the abdominal viscera, curvatures of the spine, or other deformities of the thorax. Of all these causes, those which most generally, and indeed almost exclusively, produce increased thoracic voice, are pleuritic effusions, and pneumothorax; com-

* According to Dr. C. J. B. Williams, Raciborsky, and some other authors, bronchophony may be produced by congestion of the pulmonary vessels. Now this congestion exists, in a high degree, when the mitral orifice of the heart is contracted; consequently, such contraction should always give rise to bronchophony. Congestion of the pulmonary vessels does not cause bronchophony, and most assuredly does not alter the resonance of the voice.

plete compression of large portions of the lung has very rarely been found dependent upon other causes.

It is certainly true, that, in curvatures of the spine, we sometimes find a lobe, or even an entire lung, subjected to pressure; but, nevertheless, so long as the lung is healthy, it always contains air, except in some of its smaller divisions. It rarely happens, when the abdomen is extensively enlarged, the diaphragm forced upwards, and the thoracic cavity diminished, that more than the edges of the compressed lower lobes of the lung are completely deprived of air; the upper part of these lobes almost invariably retains some portion of it.

Moreover, we scarcely ever find any considerable amount of lung-substance deprived of air by pressure, even in cases of enormous enlargement of the heart, or very extensive pericardial effusions, or large aneurisms. When the thorax contracts, in consequence of the absorption of pleuritic effusion, the lung, although much reduced in volume, always contains air, provided its parenchyma be not indurated.

The question may naturally be asked: what amount of fluid, or gas, is requisite to produce such compression of the lungs, as will give rise to the thoracic voice? Generally speaking, we cannot answer this question, for it may happen that the lobe of a lung is completely deprived of air, though not reduced to more than three-fourths of its natural size; and again, it may be reduced to a third, and even less, and still contain air. The cause of the difference is obviously this, that in the one

case, the parenchyma is loose, and contains but a small amount of fluid; and in the other, it is dense, and contains much fluid; from this circumstance, and from differences in the capacity of the thorax, we find that at one time half a pound of fluid is sufficient, while at another, several pounds are requisite, for the production of the increased thoracic voice.

If the lung be not adherent to the thoracic walls, fluids when present, always collect in the lowest parts of the pleura, thereby compressing the under portions of the lung, and very frequently giving rise to increased thoracic voice; the lower lobe of the lung, thus deprived of air, sinks beneath the fluid, by reason of its increased specific gravity; the bronchial tubes passing into it, are reduced in size, but not obliterated, nor distorted, and the air in them communicates with the air in the other bronchial tubes, provided the communication is not prevented by the presence of mucus, etc. Compression of its parenchyma does not so readily give rise to an increase in the thoracic voice in the upper, as in the lower parts of the lung; this is particularly true of the upper and anterior parts of the organ, where the bronchial tubes are more readily obliterated by compression, in consequence of the curved course which they follow. It happens occasionally, when the quantity of fluid is so considerable as to compress the whole lung, that the increased thoracic voice is heard as well marked over the anterior surface of the thorax, as beneath the scapulæ. We cannot accurately determine the distance a bronchial tube, in which con-

sonance occurs, may be from the surface of the thorax, when its consonating sounds are heard there; but no doubt the distance may be considerable. Increase of voice is occasionally met with, in cases where the amount of effusion is so great, as to produce enlargement of the thorax.

The increase of the voice is seldom produced, if the fluid is prevented from accumulating in the lower part of the thorax, and is confined to some particular spot by adhesions between the pulmonary and costal pleura. I have never observed it, in cases where partial effusions existed, about the upper parts of the lung; such effusions, however, around the lower lobe of the lung—when the fluid is present in such quantity as to cover more than the half of its surface, and so as in part, or altogether, to deprive it of air — occasionally give rise to increase in the thoracic voice.

III. THICKENING AND ENLARGEMENT (HYPERTROPHY) OF THE CARTILAGES OF THE BRONCHIAL TUBES WITHIN THE LUNGS.

The chief reason why the thoracic voice is generally louder in old than in young persons, is that in the former, the bronchial cartilages are increased in size and density. Disease may render them larger and denser than natural, both in the old and the young: but such degeneration of the cartilages (which is always accompanied by an increased, and generally purulent, secretion from the bronchial mucous membrane) is not of common occurrence, and rarely proceeds so far as to produce an increase of the thoracic voice.

Cavities in the substance of the lungs, and enlargements of the bronchial tubes—(a bronchial tube may be uniformly enlarged through the whole length of it, or its enlargement may be partial, and of a sacculated form)—do not produce increase of the thoracic voice, unless their walls reflect sound, and are infiltrated, thickened, and deprived of air, through a depth of at least several lines. A cavity, or enlarged bronchial tube, surrounded by parenchyma containing air, never gives rise to increase of the thoracic voice.

Experiments in support of the considerations offered above, respecting the causes of the variations which occur in the strength and clearness of the Thoracic Voice.

We may experiment by directing the voice into a wooden tube, fixed after death into the trachea of a person whose lung is hepatized, infiltrated with tubercle, or contains cavities; for this purpose, the lungs may be either left in the thorax, or removed entire, with the trachea and the larynx; it very seldom happens, however, that we hear the voice over the diseased parts the same as during life. In these experiments, the voice generally appears clearer in the healthy, than in the diseased parts of the lungs; and when they are taken out of the body, we find that the strength of the voice, in the normal portion of the lung, resembles pretty closely the strength of the voice, as observed during life, at those parts of the thorax beneath which the abnormal lung was situated. The results obtained are not more satisfactory, if, instead of speaking into the larynx through a

tube, we produce a sound analogous to the human voice, by blowing into the larynx through the narrowed glottis. These facts may be explained by the circumstance that, after death, fluid is almost invariably present in the bronchial tubes, the communication between the deeper bronchial tubes, or between cavities and the larynx, being generally either partially or completely interrupted by the presence of mucus, blood, serum, etc. For this reason, we are not able to obtain satisfactory results from experiments with the lungs; and it is very difficult and tedious, generally indeed impracticable, to withdraw the fluid from the bronchial tubes.

But there are other methods, by which the nature of the modifications of the thoracic voice, as they occur in the healthy and diseased conditions of the lungs, can be more readily determined. The coats of the small intestines may be considered, in respect of their power of reflecting sound, to have an analogous character to the membranous parts of the bronchial tubes; and the liver, and the substance of the heart, to resemble in a similar respect hepatized lung.

Now, if a person speaks through a stethoscope placed upon one end of a portion of intestine, moderately filled with air, the voice will be heard consonating in the air of the intestine, through a stethoscope placed upon the other end; but the force of the consonance will diminish, if its coats be much distended. If, instead of placing the stethoscope immediately upon the intestine, the auscultation be performed through the medium of a portion of liver, of lung, or of intestine filled with water, the conson-

ance will cease, or be very indistinctly heard, even though the medium employed be not more than half an inch thick, and merely large enough to close the mouth of the stethoscope.

Again, let a canal be bored along the substance of a liver, but not so as to perforate it, and then a person speak into the canal through a tube, so placed over its opening as to close it accurately; it will be found that the voice can be heard along the whole length of the canal, and to a certain distance on either side of it, considerably louder than if the person spoke through the open air; and the voice will still be heard along the course of the artificial canal, although the auscultation be performed through the medium of hepatic or pulmonary tissue several inches thick, or through bone or cartilage; it becomes weaker, however, as the thickness of the medium is increased, and at last altogether ceases.

If the liver be submerged in water, and care taken to prevent the entrance of the water into the artificial opening, the voice may be heard, even through a layer of one or two inches of water.

This experiment is more readily performed with the heart than the liver. For this purpose, the left ventricle is emptied of blood, the left auricle closed, and the aortic valves destroyed; if the voice of a person is now directed into a tube fixed in the aorta, it will be heard consonating in its cavity, through a stethoscope placed over the left ventricle; the auscultation may be practised at pleasure, through the medium of lung or liver-substance, as well as under water.

The same phenomena are also observed, when the larynx, together with the trachea and the two bronchi (which last must be closed), are taken out of the body, and a person speaks through a tube fixed in the larynx.

Again, let a portion of intestine, filled with air, be fixed under water, and two stethoscopes placed upon it, at a moderate distance from each other (care being taken that no water passes into them, which is easily managed), and then let a person speak into one of them, while the ear of the observer is applied to the other, and it will be found that the consonance of the voice in the intestine is much louder, than when the experiment is performed out of water; and that the force of the consonance is immediately diminished, if a portion of the intestine be allowed to project above the surface of the water.

These experiments seem to me to indicate pretty clearly the relation in which the increased thoracic voice stands to the different conditions of the lungs. If the consonance of the voice in the intestine, when this is not placed under water, be so feeble as to become inaudible through a medium of lung, liver, or fluid, half an inch to an inch in thickness, it seems probable that the voice in the membranous bronchial tubes will likewise be so feeble, as to become either very indistinct, or altogether inaudible, over the thorax. And again, just as the voice consonates forcibly along the artificial canal formed in the liver, in the ventricle of the heart, and in the trachea, so will it consonate forcibly in the bronchial tubes of an hepatized lung, or in the cavity of a lung infiltrated

with tubercle, and appear louder at the thorax, than the voice which passes through the open air into the ear of the observer. I have not been able to determine, by experiments on the dead body, why the thoracic voice is at one time strong and clear; at another, strong and less clear; and, in certain cases, clear and not strong.

THE TIMBRE OF THE THORACIC VOICE.

The voices of individuals, and the sounds of musical instruments, differ, not only in strength, clearness, and pitch, but (and particularly) in that quality also for which there is no common distinctive expression, but which is known as the tone, the character, or timbre of the voice.

The timbre of the thoracic, always differs from the timbre of the oral, voice, and has seldom any resemblance to that which is heard through the stethoscope, when placed upon the larynx.

The thoracic has not the roundness of the oral voice; it is generally of a tremulous character, and from this circumstance, a strong thoracic voice partakes of the timbre of the speaking-trumpet; a weak voice, of the timbre of a child's trumpet. The tremor of a weak voice is at times hardly perceptible, and its timbre then resembles that of the nasal voice; in other cases, the tremor is as strong as that produced by speaking against a paper, placed close over the teeth of a comb. The tremor is not well marked, except when the thoracic voice is clear, and its consonance increased; it is hardly to be detected, if the voice be indistinct.

It frequently happens that merely a whispering is heard in the thorax. The thoracic voice is also at times observed to take somewhat of the character of the amphoric echo, or metallic ringing sound, in cases of pneumothorax, and where large cavities are present in the lungs.

The modifications in the timbre of the voice (which admit of several degrees), may form various combinations with one another. Thus the timbre of the speaking-trumpet, as well as that of the nasal voice, and of the child's trumpet, may be accompanied by an amphoric echo, or metallic sound; and a peculiar tremulous tone be heard associated with the nasal voice. The timbre, and the strength and clearness of the thoracic voice, do not remain constantly the same in the same individual; one word may have the timbre of the speaking-trumpet, another that of the nasal voice, or of the child's trumpet, and so on.

It may be asked, where does this tremor of the thoracic voice arise? The fact of the laryngeal voice having the timbre of the speaking-trumpet, but not possessing the roundness of the oral voice, makes it probable that the voice becomes tremulous, not in the air of the bronchial tubes, but in its passage through the lung-substance and the thoracic walls. It has not been yet explained, either by clinical or post-mortem observations, why the tremor is at one time weak, and at another very strong; and why it occasionally seems to form, as it were, a second voice. This much, however, is certain, that the most marked tremor of the voice may accompany all those abnormal conditions of the lungs which give rise to in-

creased resonance of the voice; it is therefore not peculiar to any particular condition.

The fact that, in a given space, only certain sounds consonate, enables us to explain the circumstance of the thoracic voice being, in some rare cases, replaced by a whispering.

The whispering represents the articulated expiratory murmur; and when the respiratory murmur of the larynx consonates in the lungs, we hear a whispering, instead of the thoracic voice.

The causes of the amphoric echo and metallic tinkling, will be discussed hereafter.

THE PITCH OF THE CONSONATING VOICE.

The pitch of the thoracic, seems occasionally different from the pitch of the oral voice; but close attention shows us that a difference in the pitch of the consonating voice, is only to be heard accompanying the amphoric echo. We do not find that the pitch of the nasal voice, and the timbre of the speaking-trumpet, differ from that of the oral voice. I doubt whether Laennec, under the term "voix plus aigüé", understood a more acute voice (eine höhere Stimme), as it is rendered by Meissner, in his translation of Laennec's second edition.*

THE ARTICULATION OF THE CONSONATING VOICE.

The articulation of the voice is never heard distinctly in the thorax; the voice sounds as though the individual were speaking without moving the

* Laennec's Treatise, etc., translated by Meissner, pt. I, p. 56.

tongue; but, though indistinct, it is nevertheless heard in different degrees of clearness; a very strong voice appears less articulate than a weak one; and the articulation is often more marked in whispering than in speaking. The nasal voice, and the tracheal voice, are much less capable of being articulated.

LAENNEC'S DIVISION OF THE THORACIC VOICE.

Laennec distinguished:—

1. The resonance of the voice in healthy lung-tissue, and in the smaller bronchial tubes.

2. The resonance of the voice in the larger bronchial tubes, lying at the roots of the lungs, in their normal condition—Bronchophony.

3. The resonance of the voice in the bronchial tubes, when the lung-tissue is dense and consolidated —Accidental bronchophony.

4. The resonance of the voice in a cavity existing in the thorax, and containing air—Pectoriloquy.

5. The tremulous—bleating—resonance caused by fluid in the pleura—Ægophony.

I differ from Laennec, in his views respecting pectoriloquy, accidental bronchophony, and ægophony; my reasons for so doing, will appear from the following detailed considerations of these phenomena.

LAENNEC'S PECTORILOQUY AND BRONCHOPHONY.

Laennec, with the view of more accurately defining what he meant by pectoriloquy, bronchophony, &c., expresses himself thus:—"The voice passes

wholly through the stethoscope; the voice passes only in part through the stethoscope; the voice does not enter the stethoscope:" but it is manifest that whenever the voice is heard through the stethoscope, it must have passed through it. The distinction which Laennec wished to draw seems to have been this: that in one case the voice merely reaches the ear, but that, in the other, it at the same time produces a concussion in the ear. The voice is said to pass wholly through the stethoscope when it is heard, and its vibrations felt, as if it had been directed at once into the ear.

The only definition of pectoriloquy which can be gathered from Laennec's works is this, that it is the resonance of the voice in cavities; there does not appear to be any peculiarity in the voice itself which renders it characteristic of pectoriloquy. Laennec divides pectoriloquy into the perfect, the imperfect, and the doubtful. "It is perfect when it cannot be confounded with bronchophony, by reason of the distinct passage of the voice through the stethoscope, of the circumscribed nature of the phenomenon, and of the concurrent signs offered by the cough, the râles, and the respiration. It is imperfect when any of these signs are wanting, and particularly when there is no distinct passage of the voice through the stethoscope. It is doubtful when the resonance is very feeble, and in such case only to be distinguished from bronchophony by the signs observed at the spot where it occurs, as well as by the general symptoms and progress of the disease."

Bronchophony is thus defined:—"The voice rarely

traverses the stethoscope; its timbre has some resemblance to that of the speaking-trumpet; its resonance is diffused, and evidently extends to a distance. If any uncertainty still remains, it is removed by the cough, and by the sonorous inspiration which precedes and follows it: these phenomena seem to take place in lengthened tubes, and not in circumscribed spaces, and have no cavernous character."

What, according to these descriptions of pectoriloquy and bronchophony, are the signs by which they are distinguished?

The perfect passage of the voice through the stethoscope occurs only in perfect pectoriloquy; but even then, according to Laennec, pectoriloquy cannot be distinguished from bronchophony, except by taking into consideration the part where the phenomenon is observed, the extent of surface over which it is heard, and also the signs offered at the same time by the cough, the râles, and the respiration. The timbre of bronchophony is likened to that of the speaking-trumpet, but nothing is said of the timbre of pectoriloquy; we must therefore conclude that, in Laennec's opinion, no difference exists between them; had any difference existed, there could then have been no necessity for taking into consideration the situation and extent of the resonance, the nature of the cough, the respiration, &c., in order to discriminate between the two sounds.

It seems, therefore, that Laennec points out no distinctive sign between the voice, which he calls pectoriloquy, and that known as bronchophony: that the same voice is at one time called by him pecto-

riloquy, and at another bronchophony, according as it is heard in different situations, and over different extents of surface, and is accompanied by particular signs, derivable from the respiration, the râles, and the disturbance of the general functions. But surely when a voice, heard in different situations, is everywhere identically the same, it ought to have but one name applied to it.

The real question at issue is this,—how are we to ascertain, from the nature of the voice, whether the resonance heard proceeds from a cavity, or from a bronchial tube? It is not answered, simply by calling the resonance of the voice in cavities pectoriloquy, and that in the bronchial tubes bronchophony, without pointing out any essential difference between the two.

Neither have Laennec's followers, nor other physicians who have attempted the task, been more successful than himself in defining the distinction. On the contrary, most writers adduce cases in which Laennec's pectoriloquy had been heard, and yet no pulmonary cavities existed. This division of the voice into pectoriloquy and bronchophony, however, holds its ground in France, where pectoriloquy is still considered as characteristic of the presence of a cavity.

We are not able to decide with certainty by experiments on the dead body, whether the voice resounds in cavities, after the same manner as in bronchial tubes; but the reverse of this seems very improbable. We hear the voice along the course of a canal perforated in a liver,—as described above,—

just the same as we do in the experiments with the heart, and consequently as the resonance in a cavity.

If we call to mind the conditions described above as necessary for the production of the increased thoracic voice, we shall find that there is no definite sign, adapted to each particular case, which will enable us to distinguish between the resonance of the voice in cavities, and in bronchial tubes. If the size of the air-containing space were alone to be taken into consideration, a distinction might perhaps be observed; but we have shown that the force of the consonating voice is determined by the size of the air-containing space, its form, the nature of its walls, and its mode of communication with the air in the larynx; moreover, we have seen that the consonating voice is modified, by the distance of its point of origin from the thoracic walls, and by the nature of the medium through which it passes, whether lung-parenchyma, or some abnormal body.

For these reasons, I consider it certain that the perfect or imperfect passage of the voice through the stethoscope, does not enable us to decide upon the presence or absence of a cavity in the lungs; and, consequently, that attempts to draw a distinction between pectoriloquy and bronchophony are useless, and can only be productive of error.

LAENNEC'S ÆGOPHONY.

Physicians have paid even more attention to the subject of ægophony, than to that of pectoriloquy and bronchophony.

"Simple ægophony", says Laennec, "consists of

a particular resonance of the voice, accompanying or following the articulation of words. A voice, sharper and harsher than that of the patient, seems to vibrate on the surface of the lungs, resembling its echo, rather than the voice itself; it seldom passes into the stethoscope, and very rarely traverses it completely. It possesses one constant character, from which I have named it, that of trembling and hesitating, like the bleating of a goat; in its timbre, also, according to the description we have given, it resembles the voice of that animal.

"Ægophony, heard in the neighbourhood of a large bronchial tube, and in particular about the roots of the lungs, is often found mixed up with a more or less well-marked bronchophony.

"The combination of these two phenomena presents numerous varieties of sound, of which we may form a tolerably correct idea, by calling to mind the effects produced: by the transmission of the voice through a metallic speaking-trumpet, or a split reed; by a counter placed between the teeth and lips of a person while speaking; and by the nasal quivering of the voice of showmen imitating punchinello.

"The last comparison is often very perfect, particularly in persons possessing a deep voice. We frequently find that simple ægophony is present about the lower part of the external border of the scapula, in persons who present a combination of the two phenomena at the root of the lung.

"The bleating which constitutes ægophony, seems chiefly to belong to the articulation of the words, although the oral voice of the patient in no degree

partakes of it; sometimes, however, it is quite distinct, and we hear both the resonant voice and the bleating silvery echo separately, though at the same instant; so that the last would appear to arise at a point either further from, or nearer to, the ear of the observer, than the resonant voice. At times, when the patient speaks slowly, and in broken words, the bleating is heard immediately after the voice, and not with it, so as to appear like an imperfect echo at the end of the words. These two last varieties of the phenomenon in question, appear to me to occur only when the effusion is not very considerable.

"To hear the bleating distinctly, the stethoscope should be firmly applied to the thorax, and the ear laid lightly upon it; if the latter be pressed hard upon the instrument, the bleating will be much diminished in force, and the voice take more of the character of bronchophony.

"Ægophony is not confined to a distinct spot, like pectoriloquy, but is spread over a considerable extent of surface; it may generally be recognized at the same moment over the whole space between the inner border of the scapula and the vertebral column, around the lower angle of the scapula, and through a zone from one to three fingers broad, extending along the ribs, from the middle of the scapula towards the nipple.

"In a few rare cases of pleurisy, I have, in the first period of the disease, heard ægophony over the whole of the affected side; and in two of them, I ascertained after death that the phenomenon was caused by partial adhesions of the lung to the costal

pleura, preventing the lung from being pushed back towards the mediastinum, and thus causing it to be everywhere surrounded by a thin layer of serum.

"Moreover, a harsh, somewhat bleating, or reedy bronchophony (*à timbre fêlé*) is not sufficient to characterize the combination of ægophony with bronchophony, since, as we have said, ægophony is not to be considered as a distinct and certain sign, except when it reveals itself as a bleating, weak, and silvery resonance, situated at the surface of the lung."

Ægophony, according to Laennec, exists only when fluid is present in the thorax, being generally observed in cases of pleurisy and hydrothorax, where the quantity of fluid in the pleura is small. He has himself noticed ægophony in cases where the pleura did not contain more than three or four ounces of fluid; and he found that the sound always disappeared when the effusion became considerable, and especially if sufficiently abundant to cause enlargement of the thorax.

According to Laennec's views, ægophony is the natural resonance of the voice in the branches of the bronchial tubes, which are compressed and flattened by fluid in the pleura; the resonance traversing a thin tremulous layer of fluid, and becoming audible in consequence of the lung-parenchyma being compressed and denser than natural, and thus made a better conductor of sound.

In support of his opinion, Laennec offers the following considerations:—

"Ægophony is most constantly heard in those parts,—viz. the neighbourhood of the lower angle of

the scapula, and the space between the inner border of the scapula and the vertebral column, where the bronchial tubes are largest and most numerous, and where the layer of fluid effused into the pleura is thinnest when the patient is in the sitting or recumbent position.

"If the patient be placed upon his abdomen, or lie upon the opposite side to that in which the effusion is present, the ægophony is no longer heard in its ordinary situation, but becomes more distinct in some other part; it ceases to be heard altogether, if the effusion be so considerable as to compress the bronchial tubes, as well as the pulmonary tissue. But it reappears as the effusion diminishes, in consequence of the bronchial tubes, through their greater elasticity, regaining their normal state sooner than the lung-parenchyma."

Laennec endeavoured to show, by direct experiment, the influence which the intervention of fluid has in the production of the bleating sound which is characteristic of ægophony. For this purpose, he placed a bladder, half filled with water, on the interscapular region of a young man, in whom there naturally existed at this part a very clear bronchophony. The voice passing through this fluid, appeared to him, and to several other persons who assisted at the experiment, to be rendered sharper, and somewhat tremulous, though not as distinctly so as in ægophony, produced by pleuritic effusion. A similar experiment over the larynx, produced similar results.

"The bassoon and oböe, it is well known, owe

their bleating tone to the thin and flattened form of their mouth-piece, which yields to the slightest pressure of the lips, and is made to vibrate by the passage of the breath. Now, when the lung is forced back towards the vertebral column by pleuritic effusion, the bronchial tubes must become compressed and flattened, somewhat after the manner of the mouth-piece of these instruments; and thus the bronchial system of tubes becomes a kind of wind instrument, which terminates in a number of mouth-pieces, and in which the voice is resonant and tremulous. The lung-parenchyma being compressed and condensed, and thereby rendered a better conductor of sound, and the intervening fluid (a still better conductor), both assist in causing the voice to reach the ear.

"But the flattening of the bronchial tubes cannot be looked upon as the sole cause of ægophony. The extent of surface over which it is heard, and the zone of surface along which it courses, as we follow it from the under part of the scapula towards the mamma, seems to me to prove, that a thin layer of fluid, which is made to vibrate by the voice, assists much, if it be not absolutely indispensable, in the production of ægophony; and we may remark, moreover, that if simple compression of the bronchial tubes sufficed to produce ægophony, it would be constantly present in cases of contraction of the thorax, consequent upon the absorption of extensive pleuritic effusions."

Ægophony, according to Laennec, is not produced by solid exudations in the pleura, by pneumonia, by tubercular infiltrations, or by pulmonary cavities. A

combination of ægophony with bronchophony, necessarily occurs in pleuro-pneumonia, one of the sounds in turn predominating over the other. Pectoriloquy may assume the tremulous character of ægophony, but it is only in very rare cases that it does so—in those, for example, where the cavity has a flattened form, and its walls a certain degree of solidity. Lastly, ægophony, bronchophony, and pectoriloquy, may all be present together in pleuro-pneumonia, associated with abscess of the lung.

According to the preceding account, Laennec's simple ægophony is a sound of so peculiar a character, as to be always readily distinguishable from bronchophony and pectoriloquy. But, on the other hand, the modification of the voice described by him as arising out of the combination of ægophony with bronchophony, can in no way be distinguished from what he considered as simple bronchophony, having somewhat of a bleating character. How, for example, are we to distinguish the sharp, somewhat bleating, and reedy bronchophony—*à timbre fêlé*—(which does not represent any combination of ægophony with bronchophony) from the resonance of the voice, as heard in a metallic speaking-trumpet or in a split reed, or from the voice of punchinello, which last modifications of it are represented as illustrating the combinations of bronchophony with ægophony!

Whether the union of bronchophony with ægophony has really the signification ascribed to it by Laennec, whether, indeed, it be a sign of pleuro-pneumonia, but not heard in pneumonia unaccom-

panied by pleuritic effusion, are questions which Laennec himself seems to have answered in the negative; otherwise there could have been no necessity for admitting the existence of a variety of bronchophony, possessed of a bleating character, yet not ægophonic.

If, in accordance with Laennec's views, we admit that a combination of bronchophony with ægophony may exist without pleuritic effusion, it does not seem very improbable that simple ægophony may also exist without it.

It is remarkable that, while Laennec was teaching that ægophony could not exist without the presence of fluid in the pleura, many of his most distinguished followers should assert that they had observed the phenomenon in simple pneumonia. This, Laennec always attributed to their confounding ægophony with bronchophony. But what Laennec calls simple ægophony, cannot be readily confounded with simple bronchophony; and in every case in which the mistake is supposed to have occurred, there must at all events have been a combination of ægophony with bronchophony; from which it follows that this combination may exist independent of pleuritic effusion.

But as a considerable number of cases have been observed, in which there was simple ægophony without pleuritic effusion, it seems improbable that any such mistake really did occur; and consequently we cannot, despite of the great authority of Laennec, consider ægophony as a certain sign of the existence of pleuritic effusion.

Dr. C. J. B. Williams is, as far as I know, the

only writer on auscultation who entirely adopts Laennec's views on ægophony.

Dr. Reynaud, a pupil of Laennec, tells us that ægophony may be converted into bronchophony, when the patient, in whom it is heard in the erect position between the scapulæ, is made to lie upon his abdomen, or incline well forwards. The bronchophony thus produced is weak when the lung is healthy, but loud when it is hepatized. In the latter case, the moment the ægophony ceases, bronchial breathing and crepitating râles appear. From this Dr. Reynaud concludes, that ægophony is merely a remote bronchophony, that is, bronchophony heard through a layer of fluid more or less thick.

This theory has been favourably received in France, and Meriadec Laennec remarks, that Reynaud's experiments give us a sure means of invariably distinguishing ægophony proper from bronchophony, or rather, of distinguishing pleuritic effusion, either in a healthy or a hepatized condition of the lungs, from hepatization of the lungs without pleuritic effusion.

I have already spoken of Laennec's ægophony, under the name of the tremulous voice, in the chapter on the timbre of the consonating voice. I must here repeat, that I have myself met with the simple ægophony of Laennec, both when fluid existed in the pleura, and when no trace of it could be found there; also in pneumonia and in tubercular infiltrations, with or without cavities; I have also frequently found fluid in the pleura, when the consonating thoracic voice had presented neither a tremulous nor a

bleating character; and I have observed that in pleuritic effusions, as well as in pneumonia without such effusion, single words or syllables offered the tremulous bleating character of the voice, whilst other words were entirely free from it.

When a bladder filled with water is placed over the larynx of a person speaking, the voice sounds just the same as through a piece of liver of the same thickness as the depth of water in the bladder. If the experiments which have been already frequently referred to—as performed with portions of intestine filled with air, or with a perforated liver placed under water—are repeated, it will be found that the consonating voice in the intestine is not heard either bleating or tremulous through the water. I have frequently noticed the tremulous sound accidentally produced in experiments with livers, etc., both in and out of water, but I have never been able to produce it arbitrarily.

I cannot therefore, in accordance with my own experience, admit Laennec's ægophony to be a sign characteristic of pleuritic effusion; indeed this sound has been heard, both by myself and others, over the interscapular region in women and children, whose lungs were perfectly healthy.

If it be true that ægophony may exist independently of pleuritic effusion, it naturally follows that Laennec's idea of the sound being occasioned by the vibrations of a thin layer of fluid, cannot at all events be true in every case.

Without pretending to call in question the correctness of Dr. Reynaud's statements respecting the

conversion of ægophony into bronchophony by change of the patient's position, I would wish to make the following remarks thereon: Dr. Reynaud supposes that, when the patient is in an upright position, the compressed or hepatized lung is separated from the posterior walls of the thorax by a layer of fluid; but that if the patient be made to lie upon his abdomen, or lean well forward, the lung approaches the posterior walls, and the fluid gravitates towards the fore part of the thorax. Now, it is well known that hepatized or compressed lung has a greater specific gravity than pleuritic fluids; but, according to Reynaud's views, the lung must rise in the fluid, if it approach the back of the thorax when the patient lies upon his abdomen. My own belief is, that the compressed or hepatized lung of a patient lying on his back, has a decided tendency to gravitate towards the posterior part of the thorax, and that it would slightly recede from that position if he assumed an upright posture, and still more so, if he leaned forward or lay upon his abdomen. I have frequently repeated Reynaud's experiments, but have never obtained similar results.

It has occurred to me, that his observations must have been made upon patients in whom the pleuritic fluid existed in a sacculated form, and consequently was incapable of change of position. Patients suffering from pleuritic effusions not of a sacculated kind, could not endure, for more than a few seconds, the position of body necessary for carrying out Dr. Reynaud's experiments,—that is, if the effusion were present in quantity sufficient to produce increased thoracic voice.

I do not believe that flattening of the bronchial tubes has anything to do with the production of the tremulous resonance, as Laennec supposed. My own opinion is, that a tremulous sound can only be produced by the impact of one solid body upon another, or upon some fluid or aëriform body, and that mere vibrations of the air cannot give rise to it. Musical instruments, the tones of which have a tremulous character, are either tongued instruments, in which the tongue as it is called imparts impulses to the air, or instruments in which the tongue is replaced by some other contrivance.

If a disk of wood, metal, or ivory, be so placed in the mouth as to lie between the lips and the teeth, and to obstruct the passage of the air out of the mouth, it will be found that every sound excited in the larynx takes a bleating character, of uniform pitch, caused by the impact of the disk on the teeth, and bearing a most perfect resemblance to Laennec's ægophony. To the same cause, namely, impact of one solid body upon another, we must attribute the tremulous sound produced by speaking upon paper placed over the teeth of a comb.

If a person speaks into the hollow end of a stethoscope, taking care that his lips completely close the opening, but at the same time rest lightly upon it, it will be found that nearly every tone which issues from his larynx is accompanied by a tremulous sound, of uniform pitch, which arises either between the lips, or between the lips and the stethoscope.

A consideration of all these circumstances, leads me to the conclusion that Laennec's ægophony is

produced by the impact of one solid body upon another, or upon a fluid or aëriform body. Such impact, however, can only occur when the voice consonates in some air-containing space within the thorax; for, as we have already shown, vibrations are not communicated from the larynx to the parenchyma of the lungs along the walls of the trachea and the bronchial tubes. It is therefore probable that in most cases the walls of the bronchial tube, within which the air consonates, react by impact on the air contained within them, and so give rise to the tremulous sound. It is possible, however, that it may be occasionally produced by a portion of mucus, etc., partially closing the mouth of the bronchial tube, imitating the thin tongue in the mouth-piece of tongued instruments.

But whatever be the way in which ægophony is produced, it is certain that the presence of three or four ounces of fluid in the pleura alone can never produce it.

Provided ægophony does not exist in the normal state of the thoracic organs—and we have seen that it does so occasionally, in women and thin children—it will not be produced, unless the fluid in the pleura be sufficient to completely deprive of its air by compression a portion of lung, large enough to contain a cartilaginous bronchial tube.

Dr. Raciborsky gives the following explanation of ægophony:—"If the quantity of pleuritic effusion be not sufficient fully to compress the layers of air-cells, but only to force the pleura inwards upon them, so as to cause the pleura and compressed air-cells to

form, as it were, a more or less tense membrane about the extremities of the air-passages, a very peculiar character will be given to the resonance of the voice: it becomes broken and quivering, like the voice of punchinello, or like the notes of a reed-pipe. It has been compared to the bleating of a goat, and in consequence received the name of ægophony."

I am convinced that ægophony cannot arise in this way, because increased strength and clearness of the thoracic voice is only possible, when a considerable portion of lung is entirely deprived of air; and, moreover, I consider the idea of the pleura being pressed inwards on the air-cells, and rendered tense by a small quantity of fluid, to be erroneous. The lung-cells do not resist compression; they are expanded by the pressure of the atmosphere as the thorax dilates, and they likewise contract, whenever pressed upon by any body occupying the cavity of the thorax. The more the lung is distended, the more will the pulmonary pleura be stretched, and consequently pressed inwards on the walls of the air-cells. When the air-cells are diminished in size, the pleura falls into folds; and we cannot rightly speak of its exerting any strong pressure against the lung-substance, until the whole of the air is forced out of the air-cells.*

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* Fournet, *Recherches Cliniques sur l'Auscultation*, says:—"Cependant l'égophonie existe, on ne saurait en douter; et dans un certain nombre de cas, ce caractère coïncide avec un épanchement pleurétique, et sert à le faire reconnaître. Mais on peut établir en principe générale, qu'elle ne peut donner au diagnostic un caractère de certitude, qu'autant qu'elle est bornée

THE AUTHOR'S DIVISION OF THE THORACIC VOICE.

I believe I have shown that Laennec's pectoriloquy and bronchophony represent one and the same phenomenon; and that his ægophony is a sound which

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à l'un des côtés de la poitrine; qu'elle est bien nettement caractérisée; qu'elle ne cöincide point avec le caractère chévrotant de la voix auscultée à distance; qu'elle n'est point sujette, dans ses degrés, aux mêmes variations que le malade peut imprimer à sa voix; qu'elle suit les déplacements que le liquide peut éprouver dans les changements de position, auxquels on soumet le malade. Ce dernier caractère est le meilleur dont on puisse se servir, pour distinguer la bronchophonie d'avec l'égophonie; en effet le siége de la bronchophonie reste même quelles que soient les attitudes diverses que prenne le malade. Enfin l'égophonie n'a de véritable valeur comme signe d'épanchement pleurétique, qu'autant qu'elle cöincide avec d'autres phénomènes, soit locaux, soit généraux, qui de leur côté autorisent la pensée d'un épanchement dans la plèvre."

In the *Examinateur Médicale*, 15th September, 1842, there is an essay by Roger, in which mention is made of my *Treatise on Auscultation and Percussion*. After describing my views concerning the cause of the increased thoracic voice, he goes on to say:—" Le docteur Skoda n'admet que deux modifications de la resonnance de la voix, ce sont la bronchophonie, qui est forte ou faible, et un bourdonnement indistinct, qui n'a pas pour le diagnostic de valeur précise. Il ne voit pas la nécessité d'admettre, ni la pectoriloquie, ni l'égophonie de Laënnec; ou du moins, si elles existent, comme variétés de la bronchophonie, elles ne lui semblent pas mériter une dénomination spéciale. Cette réforme est certainement commode, en ce qu'elle simplifierait l'étude de l'auscultation; mais elle prive le diagnostic de ressources précieuses, et si, en effet, il est de cas nombreux de cavernes pulmonaires, et d'épanchements pleurétiques où manquent la voix caverneuse, et l'égophonie; si, d'autre part, certaines modifications, même normales de la voix, peuvent parfois

is occasionally heard accompanying the consonating voice, and which has no necessary connexion with the presence of fluid in the pleura, nor, as a sign, any especial value.

I distinguish the following modifications of the thoracic voice :—

1. The voice accompanied by a concussion in the ear, completely traverses the stethoscope—loud bronchophony, which may be either clear or dull.

2. The voice unaccompanied by concussion in the ear, passes incompletely through the stethoscope—weak bronchophony.

3. An indistinct humming (*Summen*), with or without a barely appreciable concussion in the ear.

4. Amphoric resonance, and the metallic echo of the voice. Of these, I shall speak apart hereafter.

Loud Bronchophony.

Loud Clear Bronchophony. — In this case, the thoracic voice may be either as loud as, or louder,

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simuler ces deux variétés du retentissement vocal, dans d'autres circonstances, les phénomènes morbides sont si tranchés, qu'ils ont une valeur séméiotique très-grande, surtout si on fait attention à la région du thorax où ils sont produits: une égophonie marquée est un signe à peu près certain d'épanchement pleurétique, de même que la voix caverneuse, perçue au sommet de la poitrine, annonce neuf fois sur dix une excavation tuberculeuse, et la variété que nous avons appelée voix caverneuse éteinte (v. Barth. et Roger, loc. cit. p. 185) est une signe presque infaillible de caverne tuberculeuse."

These arguments seem to me to be rather in favour of, than opposed to, my views on pectoriloquy and ægophony.

or somewhat weaker, than the laryngeal voice; the articulation being at the same time distinguishable. It indicates the presence of a considerable amount of consolidated lung-substance beneath the part of the thoracic walls where it is heard. The consolidated lung may be either immediately in contact with the walls, or separated from them by a layer of lung-tissue containing air, or by solid or fluid pleuritic exudations, provided such intervening media are of no considerable thickness. Mere fluid in the thorax does not produce loud clear bronchophony, except in the upper half of the interscapular region.

When loud clear bronchophony is heard over other parts of the thorax, we may infer the existence of one of the following abnormal states:—advanced pneumonia or pleuro-pneumonia; hepatization, with or without moderate pleuritic effusion; tubercular infiltration; pulmonary apoplexy of considerable extent; thickening of the bronchial tubes, with complete atrophy of the lung-tissue; or a high degree of pulmonary œdema, with co-existing pleuritic effusion, by which the œdematous lung has been completely deprived of its air. Of these different morbid conditions, hepatization and tubercular infiltration of the lungs are those which most frequently produce loud clear bronchophony; pulmonary apoplexy is rarely sufficiently extensive to produce it. Thickening of the bronchial tubes, with atrophy of the lung-tissue, is a somewhat more frequent cause, but still a rare one, in comparison with pneumonia and tubercular infiltration.

In cases of pulmonary œdema, when the whole of

the air is forced out of considerable portions of the lung by co-existing pleuritic effusions, or by any other cause, we hear weak, not loud, bronchophony.

The mere existence of loud clear bronchophony does not enable us to decide, whether or not cavities or enlarged bronchial tubes are present in a lung which is hepatized, or which has become indurated consequent to hepatization, or which is infiltrated with tubercular matter. But, as we know that abscesses are very rare in pneumonia, and, on the other hand, that vomicæ are rarely absent in tubercular infiltrations, we shall not often err if, in tubercular diseases, we prognosticate the presence of cavities at those parts where the voice is heard loudest; and never infer the existence of an abscess in pneumonia from the thoracic voice, however loud it may be heard.

Loud Dull Bronchophony.—The voice produces a concussion in the ear, but the articulation, and consequently the words spoken, are not recognizable.

Loud dull bronchophony is occasionally met with between the upper halves of the scapulæ of old persons, in the normal condition of their lungs, but in no other region. When heard over any other part of the thorax, it has the same signification as loud clear bronchophony.

Weak Bronchophony.

The term weak bronchophony is not used to designate mere humming, but a clear and audible voice, which produces little or no concussion in the ear; the articulation of the words uttered being

generally distinctly heard. Weak bronchophony indicates the presence of extensive pleuritic effusion and hydrothorax, in addition to the diseased conditions referred to under loud bronchophony.

Assisted by the percussion signs, we can sometimes determine upon which of these two causes bronchophony depends: if it depend upon pleuritic effusion, we find complete dulness of percussion over at least the half of one lobe of a lung, for it is necessary that the fluid should be present in such quantity, as to deprive a portion of lung, large enough to contain a cartilaginous bronchial tube, of air by compression. But if we find, at the part where the weak bronchophony is heard, that the percussion-sound is not completely dull, or not dull over so large a surface as just referred to, we may safely conclude that the bronchophony is not caused solely by fluid in the pleura, but in part by consolidated lung-parenchyma.

If, together with weak bronchophony, there is a completely dull percussion-sound over a still larger extent of surface, we cannot determine, without the aid of other signs, whether the bronchophony is a consequence of pleuritic effusion, or of hepatization of the lung. It has been said, that the doubt can be removed by changing the position of the patient. I have frequently examined patients, suffering both from acute and chronic effusions, in different positions, but have never observed any sign which could serve to solve the question.

When I am unable, either by percussion or by the character of the thoracic voice, to decide whether the phenomenon observed, proceeds from pleuritic effusion

or hepatization of the lung, I (if possible) call to aid other auscultatory signs that may be at my command, and, in particular, endeavour to ascertain the position of the neighbouring organs.

If, for example, the effusion is considerable (which it must necessarily be if the dulness of the percussion-sound be extensive), the neighbouring organs are pressed out of their natural position; but when the lung is hepatized, or infiltrated with tubercular matter—without pleuritic effusion—they almost invariably retain their normal position. If the impulse of the heart is felt at the scrobiculus cordis, and the percussion-sound is completely dull over the region of the heart and the left side, we may be sure that a considerable amount of fluid is present in the left pleura.

Loud as well as weak bronchophony passes imperceptibly into indistinct humming, which is either accompanied or not with concussion in the ear; no distinct line of demarcation can be drawn between these three degrees of the resonant voice; the extreme varieties are readily recognized, but the intermediate pass imperceptibly one into the other.

No conclusion can therefore be drawn from the resonance of the voice alone, except when it presents itself as undoubted bronchophony. If the voice be not sufficiently loud or clear to allow of positive conclusions, we may yet obtain some tolerably safe results, by comparing it at different parts of the thorax, especially at corresponding parts of the two sides. But it is always well to take into consideration every sign which auscultation and percussion

can afford us, before we come to any decided conclusion.

This precaution is particularly necessary in the investigation of the interscapular and subclavicular regions.

We are not able to draw any conclusions from bronchophony, when heard in the interscapular region, as it is often observed there in the normal condition of the respiratory organs. In the healthy state of the lungs, the thoracic voice is nowhere heard so loud and clear as over the larynx; such strength and clearness, therefore, of the consonating voice, even though heard in the interscapular region, indicates the presence of hepatized lung, or extensive pleuritic effusion. If we hear the thoracic voice as a clear whispering, *i. e.* as an articulated expiratory murmur, we may be certain that there exists some diseased condition of the respiratory organs.*

The other varieties of weak bronchophony heard in the interscapular and subclavicular regions, of themselves yield no results. A comparison of the sounds heard at corresponding parts of the two sides of the thorax must be made, and every other sign which percussion and auscultation can afford, called in to aid the diagnosis.

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* Laennec considered the clear whispering as a variety of bronchophony, and Barth and Roger looked upon it in the same light; and so also did Fournet. The clear whispering, unaccompanied by amphoric resonance or metallic tinkling, is as little a sign of the existence of a cavity, as any other vocal sound. It has the same signification as weak bronchophony. I have no doubt, that every one will recognize in the clear whispering, the phenomenon described by Fournet, vol. i, p. 159.

Indistinct Humming, with or without a barely perceptible Concussion in the Ear.

No distinct signification can be attached to the presence of this humming, nor to a complete absence of all resonance. Such humming is observed, not only in the normal state of the lungs, but may be met with in any of their diseased conditions; and the reason of this is, that bronchophony does not depend upon one, but upon many different states of the lungs. A lung may be extensively hepatized, without producing bronchophony, in consequence of the bronchial tubes of the hepatized part being filled with mucus, and not containing air.*

II. MURMURS CAUSED BY THE MOTION OF THE AIR DURING RESPIRATION.

These may be generally classed under the head of respiratory murmurs, of râles, and of whistling (Pfeifen), and sonorous sounds (Schnurren). The pure respiratory murmur is heard when the air, in its passage through the lungs, meets with no obstruction, either from the presence of fluids in the bronchial tubes, or from any peculiarity of their construction. The râles, and whistling, and sonorous sounds, are

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* Dr. Hourmann states that if the auscultator himself speak while examining a patient, he will hear pectoriloquy over cavities; bronchophony in hepatization of the lungs; and ægophony in pleuritic effusion. The experiment may be readily repeated. It will be found that bronchophony is invariably heard, whether the lung be healthy or diseased. So-called autophony is, therefore, valueless as a sign of disease.

caused by the presence of fluid in the air-passages, by thickening of the bronchial mucous membrane, and by any partial narrowing or compression of the bronchial tubes.

The Respiratory Murmur.—We know that the air produces a murmur in the mouth and nose during respiration; by placing the stethoscope over the larynx and trachea we shall also hear murmurs accompanying the respiratory movements; we are therefore fully justified in concluding that murmurs also take place during respiration throughout the whole extent of the air-passages of the lungs.

The murmur most readily heard over the thorax, would naturally be that which arises from the parts of the lung immediately beneath the surface; and as only the air-cells and finer bronchial tubes exist near the surface of the lung, it would follow that the murmur heard arises from those parts in particular. But every murmur is propagated to a distance, in proportion to its intensity; and, consequently, those murmurs also which originate in the remoter—the central—parts of the lung, in the large bronchial tubes, and even in the trachea and larynx, may be heard over every part of the thorax, in addition to the murmur of the air-cells and finer bronchial tubes.

Now if these facts be correct, the respiratory murmur as heard at any particular part of the thorax will not enable us to judge of the condition of the lung beneath it, unless we have some means of distinguishing between a near and a remote murmur—between that of the air-cells, and of the larger bronchial tubes, of the

trachea, and of the larynx; this at first sight does not appear very difficult, for we are well accustomed to judge of the distance of sounds. Our judgment may, indeed, be tolerably correct so long as the sounds pass through no other medium than the air, and their direction is not disturbed; but by auscultation it is far from easy to determine the distance and source of a sound.

By experience we obtain positive proof that a remote respiratory murmur can be heard over any part of the thorax;* thus, for example, we frequently remark a very loud respiratory murmur at parts of the thorax, beneath which there is a considerable portion of hepatized lung, that is, lung into which no air can enter. Here, then, the question meets us, How are we to distinguish between a near and a remote respiratory murmur—between the murmur of the air-cells and finer bronchial tubes, and the murmur of the large bronchial tubes, of the trachea, and of the larynx?

To answer this question it is necessary first to isolate these sounds, analyze them apart, and fix the distinctive characters which belong to each individually; secondly, to ascertain the alterations which they undergo by propagation to a distance; and thirdly, to point out those cases in which the laryngeal, the tracheal, or the bronchial murmurs, be-

* There is no doubt about the fact, although Dr. Phillip asserts the contrary.

Fournet, in the first volume of his work, p. 359, points out the changes which the respiratory murmurs undergo, when heard at a distance from their point of origin.

come consonant in the bronchial tubes, or in cavities; and moreover to show how the non-consonating tracheal murmurs, etc., may be distinguished from the consonating.

The respiratory murmur of the larynx and trachea may during life be heard isolated; and we may also isolate the murmur proper to a bronchus, by separating the tube from the trachea and the lungs, and then forcing a stream of air through it. The respiratory murmur of the air-cells and finer bronchial tubes cannot be imitated in the dead body, in consequence of fluid being constantly present in the air passages after death, so that râles are produced whenever air is forced into them: the only way by which we can obtain a knowledge of the nature of this murmur is by a comparison of it as heard in different individuals.

Now we know that, as a rule, the respiratory murmur in children is much more distinct and loud than in grown-up people, though there appears no difference in the intensity of their laryngeal murmur. There are also many circumstances which may increase the respiratory murmur in grown-up persons, and render it puerile, the laryngeal murmur remaining unaltered. We may therefore conclude that the murmur as heard in infants, presents us with the most perfect characters proper to the respiratory murmur of the air-cells and finer bronchial tubes.

The next question is, What signs have we by which to distinguish between the respiratory murmurs of the larynx, the trachea, the large bronchi, the air-cells, and the finer bronchial tubes?

Determination of the Differences in the Respiratory Murmurs.—The different respiratory murmurs may be imitated, either by drawing air into, or forcing it out of the mouth. In attempting to imitate them, it will be found that we always place the lips and tongue in the position, which is requisite for the conversion of an unarticulated into an articulated laryngeal sound; in short, that every murmur is produced by the union of a consonant and a vowel, the sound not being formed in the larynx, but in the mouth alone. That position of the mouth which is necessary for the pronunciation of any particular consonant and vowel, always gives rise to the same murmur. Hence we can distinguish sufficiently well between the different respiratory murmurs, inasmuch as they are capable of being imitated by the mouth, and as the position of the lips and the tongue requisite for the production of any murmur, can be determined by the pronunciation of a consonant and a vowel.*

The alterations which take place in the murmurs, through change of the vowels, are analogous to those which take place in the pitch of the laryngeal sounds,

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* I am well aware, that no one can obtain a just idea of auscultation, without practising it; but he who will give himself the trouble to analyze the sounds separately, will fix them with greater facility in his memory, and attain to a nearer knowledge of their peculiarities.

The respiratory murmurs are capable of very close imitation by the mouth; and the student, by frequently attempting to imitate them, will soon be convinced of the correctness of the above statement; and he will also find, that a clear idea of the distinctions drawn by me is far from useless.

particularly of the musical sounds. I shall not, therefore, trouble myself by speaking of the pitch of the different murmurs, but shall consider this determined by the vowel which is requisite for the production of the murmur. The letter *e* produces the highest pitch in a murmur, the letter *u* the lowest.

Character of the respiratory murmurs of the larynx, the trachea, and the large bronchi.—In attempting to imitate these murmurs, we find that in each case we retain the same consonant, and that whatever difference arises, depends upon a change of the vowel employed. The consonant which answers to these murmurs is *ch* guttural, or it falls between *h* and *ch*. The laryngeal, tracheal, and bronchial murmurs, may be imitated by forcing air against the hard palate; this is done involuntarily in hard breathing. The pitch of the murmur depends upon the width of the opening through which the air passes, that is, the *ch* appears in combination with different vowels. As a rule, the laryngeal murmur is higher than the respiratory murmur of the lungs.

Character of the respiratory murmur of the air-cells and finer bronchial tubes.—This murmur may be imitated by narrowing the opening of the mouth, and then drawing in the air. The consonant of this murmur is *v* or *b*. This character, however, refers only to the murmur of inspiration; that of expiration in the normal state of the respiratory organs, causes little or no sound in the air-cells and finer bronchial tubes; whatever sound is heard differs from the mur-

mur of inspiration, and resembles rather a gentle aspiration (Hauchen) or blowing (Blasen). It can only be imitated by the mouth during expiration; the consonant which represents it falls between *f* and *h*.

Changes which the respiratory murmurs undergo when propagated to a distance.—The respiratory murmurs present the above characters only when they are heard near their origin; at a distance from it, their proper characters may be lost, even though the murmur itself be of considerable strength. All murmurs and sounds lose more or less of their essential character by propagation to a distance. The rolling of a carriage is readily distinguished from the clatter of a mill, and the roaring of a waterfall from the howling of a storm, when the sounds are produced in our immediate neighbourhood; but when they reach us from a distance, their character becomes so similar, that we can no longer ascribe its particular cause to any of them.

The nature of the laryngeal respiratory murmur, as heard over the thorax through the healthy parenchyma of the lungs, may be ascertained in the following manner:—A healthy person being made to hold his breath, the observer auscultates his thorax, whilst an assistant blows into a tube, inserted as far down as conveniently may be, the throat of the person auscultated, thus exciting a loud murmur there. The murmur thus caused by the air driven through the tube, resembles the laryngeal respiratory murmur; and in consequence of its loudness and proximity to the larynx, traverses the parenchyma of the lungs as perfectly as the natural laryngeal murmur.

During the experiment a murmur may be heard, especially in the interscapular region, which does not resemble the ordinary laryngeal murmur; it is deep and hard of imitation by the mouth, and therefore its consonant is difficult to determine. The pronunciation of the consonant *f* during expiration gives us the best idea of it.

It happens occasionally, when pleuritic effusion has so thoroughly compressed a lung as to prevent all entrance of air into it, that a respiratory murmur is still heard over the affected side, particularly in the space between the scapula and the spine, and beneath the clavicles; such murmur must of necessity have its origin in the trachea or one of the large bronchial tubes, and hence we have means of judging what changes the tracheal murmur undergoes by its passage through a considerable amount of liquid. It becomes deep, and no longer resembles the ordinary tracheal murmur; if anyone attempt to imitate it by the mouth, he will find it best represented by the consonant *f* pronounced during expiration.

We can judge of the character of the respiratory murmur of the air-cells and finer bronchial tubes, by auscultating over those parts of the thorax beneath which we have ascertained by percussion that there is no lung present, but which are not so far from the lung as to prevent our hearing the murmur. In this way we shall find, that by distance the murmur loses its sipping character, and is converted into something between an aspiration and a blowing, very difficult of imitation, and also best represented by the consonant *f* pronounced during expiration.

In certain perfectly healthy individuals, the inspiratory murmur is scarcely audible, or, at all events, has not the characters which are here given as proper to it, although in these cases the air assuredly enters into the air-cells of the lungs: it resembles rather a remote murmur of the air-cells or of the trachea, but has nothing of a sipping (Schlürfen) or gasping (Keuchen) character, and cannot therefore be safely referred to either the one or the other of these sounds.

Conditions under which an increase by consonance of the laryngeal, tracheal, and bronchial murmurs takes place within the lungs; and the difference between the consonating and non-consonating tracheal murmurs as heard over the thorax.—If it be true, that the thoracic voice is increased by consonance in certain altered conditions of the respiratory organs, there can be no doubt that the laryngeal, tracheal, and bronchial respiratory murmurs may likewise be increased by consonance within the thorax, and be heard louder and clearer than natural over the surface of the thorax. It is evident, that the conditions necessary to produce consonance of the respiratory murmurs, are the same as those required for the voice; I shall not, therefore, repeat them here.

The distinction between the consonating and the non-consonating respiratory murmurs, or murmurs merely conducted from a distance (as heard over the thorax), becomes evident, if we consider the difference between the consonating and non-consonating voice. If the conditions necessary for consonance

are present, the voice is heard as such over the thorax, otherwise it is recognized only as a humming. In like manner, the consonating laryngeal murmur is heard as a laryngeal murmur over the thorax, except in cases of large excavations or of pneumothorax, where the amphoric resonance or metallic tinkling is associated with the murmur: the non-consonating laryngeal murmur has the characters already described.

LAENNEC'S DIVISION OF THE RESPIRATORY MURMURS.

1. Pulmonary—Vesicular—Respiratory Murmur.
2. Bronchial Respiration.
3. Cavernous Respiration.
4. Blowing Respiration, and Masked Respiration (*souffle voilé*).

Laennec's Vesicular Respiration.

This he describes as a weak but very distinct murmur, audible during inspiration and expiration, indicating the entrance of air into the pulmonary tissue, and its consequent distension. The laryngeal, the tracheal, and bronchial murmurs, he tells us, are audible only in the interscapular space and behind the sternum, and even there almost wholly masked by the vesicular murmur. He draws no distinction between the inspiratory and expiratory murmurs, and appears to set down every thoracic murmur which is not bronchial, cavernous, blowing, or amphoric, as vesicular.

I believe that this view is incorrect, for the reasons I have given above, and consequently that the signi-

fication attached by Laennec to his vesicular murmur, cannot be true in all cases.

"When the respiration", says Laennec, "is distinctly heard of pretty equal strength over every part of the thorax, we may be sure that neither pleuritic effusion nor engorgement of the pulmonary tissue has taken place; but, on the contrary, when we find the inspiration deficient at a certain part, we may safely conclude that the subjacent lung is impermeable to air; this sign is as characteristic, and as readily distinguished, as the existence or absence of the proper percussion-sound of the part (as ascertained by the method of Auenbrugger), and supplies precisely the same indications. Absence of sound is always associated with absence of respiration, excepting in some few cases, in which a comparison of the results obtained by auscultation and percussion will afford signs which are perfectly pathognomonic.

"Auscultation, as we shall see, has the advantage of indicating the different degrees of pulmonary congestion with greater certainty than percussion; it has the disadvantage of occupying more time, but its practice requires less care and attention than percussion; it is available in every case, and even serviceable in those which yield no results to the method of Auenbrugger."

Now I am convinced, that the respiratory murmur may be heard of equal strength and clearness over the whole of the thorax, even when considerable portions of the lung are distended and congested, and that it may be strong at one part, and weak and indistinct at another, when there exists no abnormal

affection of the lungs which is appreciable. I therefore dissent from the opinion, that auscultation is a surer indication than percussion of the different degrees of pulmonary congestion; I hold it essential, in every case, to take into consideration the percussion signs and the general symptoms, as well as the indications obtainable by auscultation.

Laennec's Bronchial Respiratory Murmur.

By this murmur, Laennec understood the sound produced during respiration by motion of the air in the larynx, the trachea, the large bronchial tubes at the root of the lung, and in the smaller bronchial tubes. In the healthy condition of the respiratory organs, however, it is not heard in the smaller bronchial tubes, being mixed up with the vesicular murmur. Hence the bronchial murmur is audible only in the neighbourhood of the larynx, and occasionally over the surface of the neck; in certain (and particularly so in thin) persons, the murmur heard behind the sternum and in the interscapular region, partakes of the character of the bronchial murmur, but is hard to be distinguished, being commingled with the vesicular murmur.

Should the pulmonary tissue have become hardened or condensed from any cause, as from pleuritic effusion, peripneumonia, or pulmonary apoplexy, and the respiratory murmur be notably diminished, or have entirely disappeared, we then often distinctly hear the bronchial respiration, not only in the larger bronchial tubes, but also in the smaller branches. This phenomenon Laennec explains in the following

manner :—" The entrance of air into the air-cells is prevented by the congestion and condensation of the pulmonary tissue, and hence bronchial respiration alone takes place, and it is heard so much the louder and more readily, in consequence of the sound-conducting power of the tissue being increased by its condensation.

The bronchial respiration in such abnormal states of the lung, is heard with most distinctness about the root and apex of the lung ; and the reason of this is, that at the root the bronchial tubes are widest, and at the apex most subject to dilatation.

I do not consider Laennec's opinion of the cause of the bronchial respiration correct. The bronchial respiration is often heard remarkably loud in cases where the lung-tissue is compressed, or completely hepatized, so that no air can enter into it; but an increased current of air would of necessity be required to produce the bronchial murmur, if Laennec's views were correct. Now, during inspiration, the air rushes into the lung with greater force and rapidity, the greater the power of expansion of that organ ; and is more forcibly driven out again during expiration, the more firmly the lung contracts, or is compressed. The less any part of a lung expands during inspiration, and contracts during expiration, the less will be the current of air flowing into or out of the bronchial tubes ; when the lung-tissue is completely compressed or hepatized, the current will be almost nil. As no change of volume takes place in a hepatized lung during respiration, we can hardly speak of any current of air flowing into or out of it.

The slight contraction which the bronchial tubes possibly undergo during expiration from pressure of the thoracic walls, and the equally slight expansion during inspiration, may certainly permit the entrance of air into them, but can cause no current capable of producing the loud bronchial murmurs frequently heard over the thorax.*

For similar reasons, I cannot adopt Andral's explanation of bronchial respiration. It is as follows: "When there is any obstruction to the entrance of air into the air-cells, the pressure of the air upon the bronchial tubes is increased, and, consequently, a louder murmur is produced within them." My belief is, that the bronchial respiration must be explained by the laws of consonance. When, for instance, the walls of any large pulmonary cavity, or of the bronchial tubes, which run into the parenchyma of the lungs, are solid enough to reflect sound, the respiratory murmur of the larynx, of the trachea, and of the two bronchi, will consonate in the air contained within them.

Laennec's Cavernous Respiration.

"By the term cavernous respiration," says Laennec, "I understand the murmur which occurs during inspiration and expiration in a cavity formed in pulmonary tissue, either through softening of tubercular matter, or through gangrene, or through abscess

* Fournet and Barth and Roger, all give the same explanation of the bronchial murmur as Laennec does; but they state, in addition, that the thickened pulmonary tissue consonates and strengthens the sound.

consequent on pneumonia. This kind of respiratory murmur has the same character as the bronchial respiration, but the air seems to penetrate into a larger cavity than that of a bronchial tube. If any doubt as to its nature arise, it may readily be removed by observation of the resonance of the voice, and of the cough."

It is not possible from such a description to draw any distinction between bronchial and cavernous breathing. Daily experience teaches us that various kinds of murmurs are produced by air, when it enters into a wide and confined space: of such, those to which Laennec gave the name of amphoric echo, and metallic tinkling, can alone be considered as characteristic of a cavity. This, however, does not appear to have been Laennec's idea, for he speaks of the amphoric echo as heard during respiration, and of its signification, under another head altogether.

If Laennec had been able to observe any constant distinctive sign between cavernous and bronchial respiration, he would doubtlessly have endeavoured to give an idea of it by comparison with some well-known murmur, and not have proposed *that* as a sign of cavernous breathing, which cannot possibly be a sign.

The statement made by Laennec, viz., that air is heard entering into a cavity larger than that of a bronchial tube, furnishes us with no characteristic sign, for what the peculiarity of the murmur is which indicates the entrance of air into the larger space, is not defined.

A cavity in a lung does not always yield the same murmur: the murmur varies according to the size of the cavity, the number and diameter of the bronchial tubes which open into it, and the condition of its walls. The murmur is also more or less distinct, according as the cavity is more or less distant from the walls of the thorax. Some cavities in the lungs have such firm and hardened walls, that any diminution or enlargement of them during respiration must be very slight, and in some cases impossible; consequently, no air passes into them during inspiration, nor out during expiration; yet, if they contain air, and communicate with the bronchial tubes, a very loud murmur is generally produced within them during respiration: this murmur is evidently an effect of consonance. The air in the cavity vibrates in unison with the air contained in the nearest bronchial tube which communicates with it, and in which a murmur is produced by the current of air during respiration; the murmur thus excited in a cavity by consonance is bronchial, excepting when the cavity is of sufficient magnitude to give rise to amphoric echo or metallic tinkling.

Some cavities, though their walls are solid, are capable of being compressed, and consequently admit air during inspiration, and expel it during expiration. According to the laws of consonance, we ought in such cavities to hear both the respiratory murmur of the nearest or even of distant bronchial tubes, and that also which is caused by the current of air flowing into and out of the cavity, provided the change in its capacity be sufficiently great. The

murmur, however, produced by consonance, is always either bronchial or amphoric; and that excited by the passage of the air into and out of the cavity, must, owing to the solidity of its walls, have the same character, provided it be not accidentally converted into a hissing, whistling, or sonorous sound, in consequence of the narrowness of the mouth of the bronchial tube which opens into the cavity.

Little or no consonance occurs in cavities, the walls of which are merely membranous, and immediately surrounded by air-containing lung-tissue; neither are bronchial breathing, amphoric echo, nor metallic tinkling, produced within them. The air streaming in and out (provided the communication be sufficiently free) causes a feeble murmur, not resembling the vesicular murmur, but something between an aspiration and a blowing: when the communication is narrow, whistling, hissing, and sonorous sounds arise. If the cavity is large, and the opening into it narrow, and particularly if there are several such cavities, we hear, in cases of dyspnœa, a loud hissing during inspiration, followed by one or more clicks at the end of inspiration,—a sound resembling that produced by suddenly stretching a slip of paper, and which is known under the name of craquement, dry crepitant râle.

Laennec's Blowing Respiration (*respiration soufflante*).

When the respiratory murmur produces in the ear of the observer a sensation as of air being drawn from his ear during inspiration, and blown into it during expiration, it gives us a representation of

Laennec's blowing-respiration; this blowing, according to him, accompanies bronchial and cavernous breathing only, and occurs in those cases in which the bronchial tubes or the cavity lie very near to the thoracic walls. The vesicular respiration may produce a similar illusion, when the inspiratory and expiratory murmurs are sufficiently strong. The bronchial murmur does not become blowing, unless its intensity is increased.

But the intensity of the respiratory murmur does not depend solely upon the distance of the bronchial tubes, or the cavity in which the murmur arises from the thoracic walls; it depends also upon the rapidity and extent of the respiratory movements, and upon the force of the consonance. The blowing bronchial respiration does not invariably indicate the presence of a cavity, or of a bronchial tube lying immediately subjacent to the thoracic walls.

Laennec speaks of another modification of the blowing respiration, as the masked blowing (*souffle voilé*), in the following terms: "It seems as though every vibration of the voice, the cough, and the respiration, puts in motion a kind of moveable veil, placed between the cavity and the ear of the observer. The phenomenon is observed—1. In tubercular cavities, the walls of which are at some points very thin, and at the same time soft and flexible, having no attachment, or but a very slight one, to the walls of the thorax; 2. In cases where the walls of peripneumonic abscesses are in a state of unequal inflammatory induration, being still at some points in the stage of congestion; 3. The phenomenon is

still more frequently observed in cases where bronchophony, produced by pneumonia in some of the large bronchial trunks, is present, the affected bronchus being in some parts of its course surrounded by healthy pulmonary tissue, or tissue but slightly congested, which is placed between the bronchus and the ear of the observer; 4. Dilatation of the bronchial tubes and pleurisy are sometimes attended by the same phenomenon, under analogous circumstances,—that is, when the cavity in which the resonance of the respiration, of the voice, or of the cough takes place, has its walls much less dense in some parts than in others.

"This phenomenon must not be confounded with the large bubbling mucous râle which sometimes attends it; the difference is readily recognized by any one accustomed to auscultation."

It is impossible to understand exactly the nature of the murmur, to which Laennec applies the term *souffle voilé*, for in his description of it, he does not give us the means of comparing it with any known murmur. In works on auscultation which have appeared since Laennec's time, the souffle voilé is scarcely referred to. I have never myself met with any murmur which is constantly associated with the conditions described by Laennec as requisite for the production of the souffle voilé, and which is observable in no other case. I believe that Laennec, by souffle voilé, intended to represent the phenomenon which is observed when the respiratory murmur is indistinct at the commencement of inspiration, but suddenly becomes bronchial, and even bronchial blowing, as the inspiration advances; the expiratory murmur, on

the other hand, being loud at its commencement, and indistinct at its conclusion.

Such a modification of the bronchial breathing indicates nothing more than this: that at the commencement of inspiration and at the end of expiration, the communication of the bronchial tube, or the cavity whence the bronchial breathing proceeds, with the other bronchial tubes, is either partially or completely interrupted, and is restored again during inspiration.

THE AUTHOR'S DIVISION OF THE RESPIRATORY MURMURS.

I consider Laennec's bronchial and cavernous respiration to be one and the same murmur; his blowing bronchial to be a loud bronchial murmur; and his souffle voilé to be an unimportant modification of the bronchial respiration. I am also convinced that there are respiratory murmurs audible over the thorax, which cannot be classed either under the head of bronchial or of vesicular murmurs.

The following is my division of the respiratory murmurs:—

1. The pulmonary respiratory murmur, or vesicular breathing of Andral.
2. Bronchial breathing.
3. Amphoric echo and metallic tinkling heard during breathing; of these I shall speak hereafter.
4. Indeterminate (*unbestimmte*) respiratory murmurs.*

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* Dr. Phillip, following Fournet, says:—"Metallic tinkling, amphoric echo, the cavernous, the bronchial, the blowing, the

Vesicular Breathing.

By vesicular breathing, I understand that murmur only which is heard during inspiration; it resembles the sound made by sucking in air through the lips. I do not apply the term to any inspiratory murmur which does not distinctly offer this character, even

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resonant respiratory, and the clear respiratory murmur—all come under the same type, the metallic. In many diseases—as in phthisis—we see them passing through every shade, one into the other. There is no change in the nature, the essence of the murmur, but merely a gradual conversion of one variety into another."

But what is the essence of the murmur? I can scarcely believe that any one will, upon reflection, repeat this indefinite description of Fournet. What do we gain by being told that metallic tinkling, cavernous and bronchial breathing, etc., are not distinct murmurs, but merely degrees of the same murmur? The question still remains: What are the conditions indicated by these different murmurs, or these different degrees of the same murmur? In this respect, amphoric echo and metallic tinkling must be separated from all the varieties of bronchial breathing. If the gradual passage of one murmur into another justified us in classing them under one head. then we may say that all murmurs must be classed together, for all pass gradually one into the other.

But Fournet, notwithstanding his assertion of similitude of type between these murmurs, could not bring himself to place the cavernous and the bronchial breathing in the same class. He says:—"Ce n'est pas que, lors de l'absence des caractères caverneux et amphoriques, on doive conclure nécessairement à la non-existence de cavernes; car, il arrive quelquefois que, dans certaines dispositions anatomiques des parties malades, le caractère bronchique au deuxième, ou troisième degré, qui se passe dans une partie rapprochée de l'oreille, obscurcit, ou même efface tout-à-fait le caractère caverneux à un faible degré qui

though it be the inspiratory murmur of a perfectly healthy individual. I am convinced that a respiratory murmur of the character here given, can only be produced by the passage of the air into the air-cells of the lungs. The expiratory murmur has no relation whatever to vesicular breathing; it may be weak or strong, or altogether absent, without in any way affecting the vesicular breathing.

I explain the sound of the vesicular breathing as Laennec does, attributing it to the friction of the air against the walls of the finer bronchial tubes and the air-cells, the contractile power of which it has to overcome. The reason why the inspiratory murmur of the air-cells is much louder than the expiratory is, that the air, when it enters into them, meets with resistance from their contractility, but does not meet with any in its passage out of them. It is otherwise, however, with the large bronchial tubes, and particularly with the trachea and larynx; here the air, during inspiration, meets with no opposition —it has, indeed, rather a tendency to expansion; but during expiration, being forced from a larger—the air-cells—into a smaller space, it is compressed; and hence the expiratory murmur of the larynx, the trachea, and the large bronchi, is, as a rule, louder than the inspiratory.

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se produit dans les parties centrales du poumon."—*Recherches cliniques sur l'Auscultation*, pt. ii, p. 519.

Barth and Roger entirely follow Laennec, in regard to the significations of the respiratory murmurs. I believe that I differ somewhat from the generally received opinion, in representing the bronchial and cavernous respiration as identical.

This fact of itself almost suffices to disprove Beau's theory of the cause of vesicular breathing; his explanation is this:—the sound produced by the impulse of the air against the soft palate, and the neighbouring parts, is propagated along the whole length of the columns of air contained in the tubes. I believe I have already shown that the respiratory murmur of the soft palate, the larynx, the trachea, and the large bronchi, is never heard as vesicular breathing over the thorax when the lungs are healthy; in certain diseased conditions of these organs it is sometimes heard as bronchial breathing. Beau appears, in his explanation of the respiratory murmur, to have been guided by observation of the bronchial breathing heard over the thorax.

The presence of the vesicular murmur at any part of the thorax, indicates the entrance of air into the air-cells of that part of the lung which lies beneath the spot auscultated. Its existence, therefore, excludes those abnormal conditions which prevent the passage of air into the air-cells; such, for example, as their compression by exudations, or tumours in the pleura, by enlargement of the heart, and other diseases; infiltration of the lung-tissue by plastic or tubercular matters, by blood, serum, etc., atrophy of the air-cells, and the obliteration of the bronchial tubes by mucus and blood, or by swelling of the mucous membranes.

Solitary tubercles, however abundant, do not necessarily interfere with the vesicular respiration, nor does inflammation confined to a few lobules—lobular hepatization;—the vesicular murmur is, in fact, fre-

quently observed in the course of these diseased conditions of the lungs.

The greater the opposition produced by contractility, to the entrance of air into the air-cells, and the more rapid and complete the inspiratory movements, the louder will the vesicular breathing be. The force of the vesicular respiration is also modified by the different conditions of the lining membrane of the air-cells and the finer bronchial tubes; it always becomes louder when of a coarse character—the coarseness indicates a slight degree of swelling of the mucous membrane of the finer bronchial tubes, and the air-cells.*

Vesicular breathing may pass gradually into the indeterminate respiratory murmur, and harsh vesicular breathing into hissing, whistling, and sonorous sounds.

The tone of the vesicular is almost always deeper

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* Fournet treats of the qualities of respiratory murmurs under the following heads: 1, their essential character; 2, their character of hardness and softness; 3, their character of dryness and moistness; 4, their timbre; 5, pitch; 6, intensity; 7, duration; and 8, rhythm. I believe, that the timbre of the murmur is its essential character. Fournet himself says:—"Le caractère propre de la grande classe des altérations de timbre consiste dans cette impression auditive, que l'on a rendue par le nom de métallique"; that is, the metallic timbre forms the essential character of a certain class of sounds. The character of hardness is better interpreted by the term of harshness; the healthy vesicular murmur is gentle and soft, quite unlike the coarse vesicular murmur. The term moist is not applicable to the respiratory murmur; we cannot speak of a moist or dry respiration, the idea of moistness can be attached only to râles.

than that of the laryngeal respiratory murmur. It is sometimes inordinately acute, particularly in the upper parts of the lungs, in old people, and in cases of pulmonory œdema, and when solitary tubercles are thickly scattered through the tissue of the lungs; in some rare cases it is even more acute than the laryngeal murmur. An acute vesicular murmur of this character is nearly allied to a hissing sound. The vesicular breathing may be unattended by an expiratory murmur; when the latter is present, it varies considerably in strength, and is occasionally much louder than the inspiratory. The presence of an expiratory murmur always indicates the existence in the bronchial tubes, of some obstruction to the egress of the air from the lungs. The impediment is, in most cases, caused by a swelling of the lining membrane of the bronchial tubes. The expiratory murmur is, with very few exceptions, of a deeper tone than the inspiratory; and the depth is in the ratio of the distance of the bronchial tube, in which it arises, from the surface of the lung. It approaches the pitch of the vesicular inspiration in those cases only, where the current of air meets with obstruction in the finest bronchial tubes. Fournet has carefully investigated the relation of the expiratory to the inspiratory murmur, and he has expressed their strength and duration by figures, in order more accurately to define their relative degrees.

The normal inspiratory murmur, that is, vesicular breathing, may be expressed by the number 10, and normal expiration by 2; in other words, the strength and duration of the latter are only one fifth of those of the former.

Under abnormal circumstances, inspiration may fall from 10 to 0; the expiratory murmur either sinking in a like proportion, or remaining unchanged, or rising even as high as 20. The intensity of the inspiratory may also increase, whilst that of the expiratory murmur either remains unaltered, or is also increased. Lastly, the intensity alone of both the inspiratory and the expiratory murmur, may be either increased or diminished, the duration either remaining unaltered, or increasing or diminishing in different proportions. Their duration may also differ from the normal standard, without the intensity undergoing any alteration.

Fournet considers that increased intensity of the expiratory, attended by diminution in that of the inspiratory murmur, is a very important diagnostic sign; the higher degrees of such abnormal changes being observable in cases of emphysema and tubercle of the lungs; such variations in the intensity of the murmurs also occur, but in a less marked degree, in cases of induration of the lung from other causes, in acute catarrh, and in pleurisy, accompanied by moderate effusion. I am, nevertheless, still of opinion that an increased expiratory murmur—provided it has not a bronchial, nor any other character than that proper to it—indicates nothing more than this: that the air, in passing out of the lungs, meets with some obstruction in the bronchial tubes.

In some rare cases, the acute development of solitary tubercles produces no alteration whatever in the respiratory murmur; most generally, however, it is attended by signs of catarrh. The slow formation of

tubercle in isolated spots, either produces no signs at all, or, if any, those of catarrh or of pleurisy.

A marked prolongation of the expiratory, accompanied by a shortened inspiratory murmur, is not observed in chronic development of tubercle, unless the tubercular mass be considerable, or cavities be also present in the lungs. Whenever there is extensive condensation of the lung-tissue, from any other cause, the prolonged expiratory, and shortened inspiratory murmurs always co-exist. In such cases, however, the strength of the expiratory murmur is frequently produced by consonance; and it often happens, that an indistinct expiratory murmur is converted by a deep inspiration into bronchial breathing, and thus is frequently associated with bronchophony. The intensity of such an expiratory murmur occasionally indicates its approaching conversion into a sonorous sound, or of its having already assumed the character of a weak sonorous sound.

The murmurs attending emphysema of the lungs vary much, according to the extent of the disease, and the amount of catarrh accompanying it. The expiratory murmur is very slight, when the bronchial tubes contain no mucus, and are not swollen; but it becomes full and prolonged, if their mucous membrane be thickened, or the bronchial tubes in any way narrowed, as by contraction: generally speaking, the murmur ceases to be a respiratory murmur, properly so called, but is converted into a sonorous, a whistling, or hissing sound,—in short, emphysema of the lungs yields the same auscultatory signs as catarrh.

Bronchial Respiration.

A respiratory murmur cannot be called bronchial, unless it has the character of a laryngeal or tracheal murmur, differing from the latter only in respect of its pitch. Bronchial breathing may be imitated by blowing into a tube; to imitate it with the mouth, the tongue must be so placed as to form the consonant *ch*—guttural—and the air then drawn in and forced out. The bronchial respiration heard over the thorax may be higher, stronger, deeper, or weaker than the laryngeal murmur, or it may be of the same strength and pitch; the reason of its thus varying in character is, that the bronchial respiration is not always a consonating laryngeal respiratory murmur, but frequently proceeds from the lower part of the trachea, or from one of the bronchial trunks, or even from one of the large bronchial branches. The different degrees of strength and pitch of the bronchial respiration heard over the thorax, afford no special indication, inasmuch as such modifications are not produced by one, but by several causes.

The different degrees of strength and pitch of the respiratory murmur of the larynx, the trachea, and the bronchi—which depend in part upon the rapidity and extent of the respiratory movements, and in part upon the condition of the lining membrane of the air-passages, and the more or less perfect consonance of the murmur within the diseased lung (which takes place after the manner I have described)—produce changes in the strength and pitch of the bronchial respiration heard over the thorax.

The bronchial respiration is generally heard louder over the thorax during expiration, than during inspiration, in consequence of the tracheal, the laryngeal, and bronchial expiratory murmurs being for the most part louder than the inspiratory; but there are exceptions to this, for the inspiratory murmur may be the louder, or exist alone;* or the expiratory only may be heard; or the inspiration may commence with an indistinct vesicular, and then pass into a bronchial murmur.

These modifications are all accidental; they are caused for the most part by the interruption (through the presence of mucus, blood, etc.) of the communication between the bronchial tubes, and may vary from minute to minute.†

Bronchial respiration offers the same indications as weak bronchophony, and I therefore refer the reader to what has been already said under that head;‡ it

* "Jamais je n'ai vu le caractère bronchique normale ou morbide, exister pendant l'inspiration seulement," says Fournet, p. 58.

† Barth and Roger, p. 83, observe:—"Du reste, le phénomène—la respiration bronchique—est continu, permanent, et n'est point sujet à des intermittences." I must entirely deny this assertion. The bronchial respiration appears and disappears, and is replaced by other murmurs.

‡ Barth and Roger, p. 88, assert, that bronchial respiration very rarely occurs, in cases where effusion exists alone, without thickening of the lung-parenchyma; it occurs, they say, first, occasionally at the commencement of an attack of pleurisy, when the quantity of effusion is small, and the respiration strong; and secondly, in cases where the effusion is spread equally over the lung, in consequence of its being partially attached to the walls of the thorax. Except in these cases,

does not however occur, like weak bronchophony, in the normal state of the respiratory organs. It may be heard occasionally, in healthy persons, about the upper dorsal vertebræ, and, in cases of great dyspnœa, between the scapulæ, and sometimes over every part of the thorax, without there being any change of structure in the corresponding portion of lung-tissue. This anomaly may be explained by the circumstance, that when the respiratory murmur of the large bronchial tubes is loud at its origin, it may become audible as bronchial respiration over the thorax, without being strengthened by consonance.

Bronchial breathing may pass gradually into the intermediate respiratory murmur, into amphoric echo and metallic tinkling, and into consonating whistling, hissing, and sonorous sounds.

Indeterminate (*unbestimmte*) Respiratory Murmur.

Under this title I include such respiratory murmurs as have neither the character of vesicular nor of bronchial respiration, are not attended by amphoric echo nor metallic tinkling, nor are represented by

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bronchial respiration invariably indicates condensation of the lung-tissue.

There is no doubt that bronchial respiration is most frequently heard in cases of hepatization and tubercular infiltrations of the lung; but it is also heard when effusion alone exists, not indeed under the circumstances stated by Barth and Roger, but in every case where the conditions necessary for the production of consonance present themselves. Equally incorrect is the statement, that the bronchial respiration caused by effusion, is distinguishable from that caused by hepatization of the lung.

any of the other murmurs hereafter to be described, which accompany respiration; viz., whistling, hissing, and sonorous sounds, or pleural friction sounds.

The respiratory murmur of the air-cells is sometimes so little marked, as to be in no way distinguishable from the murmur which proceeds from the deeper bronchial tubes, or even from the larynx, and which is propagated, unaided by consonance, through the parenchyma of the lungs, to the thoracic walls. A weak and remote râle may also be heard over the thorax, as an ill-defined respiratory murmur of the air-cells. No distinct indication can in any particular case be drawn from such a murmur, as several causes may concur in producing it. We cannot ascribe it with any certainty either to the entrance of air into the air-cells, or to the passage of air through the larger bronchial tubes, or to a weak and remote râle, though we may be certain that it depends upon some one or other of these causes, or upon some combination of them.

Moreover, the murmur of the larger bronchial tubes may, without either consonating or partaking of the character of the bronchial respiration, be heard so distinctly over the thorax, as at once to satisfy the observer that it is not formed in the air-cells; but in such a case we cannot tell whether the air does or does not enter into the air-cells, for either alternative is possible; and hence, from a murmur of this kind, no conclusion can be drawn as to the state of the pulmonary parenchyma; neither does the expiratory murmur, provided it be not bronchial, nor amphoric, afford us any indication of the condition of the lung.

All respiratory murmurs which give us no information as to the state of the parenchyma of the lungs, I call indeterminate respiratory murmurs—any subdivision of them appears to me useless.

A loud indeterminate respiratory murmur indicates that obstruction exists to the passage of the air in some of the bronchial tubes; its strength and pitch enable us to judge tolerably well of the size of the tubes. The indeterminate respiratory murmur passes gradually into whistling, hissing, and sonorous sounds.

I have not troubled myself to describe accurately the characters of the respiratory murmurs; for I do not consider that there is any great difficulty in distinguishing a murmur, provided it be not one of those sounds which characterize the transition of one murmur into another. The more delicate and practised the ear of the observer, the more readily will he judge rightly of these transition-murmurs; it is, however, much the safer plan to regard all respiratory murmurs which are not distinct in character, as indeterminate murmurs, and to draw no conclusion from them without due consideration of all the other indications obtainable by percussion, etc. With such precautions, an observer, though but moderately practised in auscultation, will rarely fail in his object.*

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* Fournet, vol. i, p. 82, gives the following description of a peculiar respiratory murmur:—"On apprécie très bien par l'auscultation, à la sensation reçue par l'oreille, si l'air pénètre bien avant dans le tissu pulmonaire, et à quel degré se fait l'expansion de ce tissu. Si un épanchement pleurétique considérable

RÂLES.

Causes of the Râles and their Varieties.

The râles heard during respiration are, for the most part, caused by the passage of air through liquids, such as mucus, blood, serum, etc., which are accidentally present in the bronchial tubes, or in pulmonary cavities. A kind of râle may also be produced by solid bodies—as, for example, by a fold of mucous membrane—when they partially obstruct the air, and are made to vibrate. A peculiar râle is also produced when a current of air passes into a lung which is distensible, but has lost its contractile power.

The respiratory murmurs may be either completely masked by the râles, or heard in concert with them.

Râles resemble the bubbling of boiling water or

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comprime le poumon, on sent, en quelque sorte que l'air, après avoir pénétré un peu dans l'arbre bronchique, ne peut pas aller plus loin ; on sent qu'alors il lutte un instant contre l'obstacle qui s'oppose à la dilatation des vésicules, et que, ne pouvant pénétrer dans celle-ci, il fait exécuter au tissu pulmonaire comprimé, une sorte de dilatation en masse qui s'accompagne d'un petit bruit tout particulier. Il n'est pas de mot, qui puisse rendre exactement cette sensation ; il faut l'avoir éprouvée ; mais il importe beaucoup d'en marquer le degré, afin de savoir où l'on en est de la marche de la maladie", etc.

The statement that the air struggles for an instant against an obstacle, and then, because the air-cells will not allow of expansion, dilates the lung-substance in mass, hardly needs refutation. Air enters only where a vacuum is formed, and does not struggle against obstacles, except when a vacuum exists beyond them. I am unacquainted with the murmur described by Fournet; it is impossible to follow a statement so mixed up with hypothesis.

of fat, the breaking of bubbles on the surface of fermenting fluids, the fine crackling of the little bubbles which rise to the surface of water beginning to boil, or of roasting fat, the crackling of salt on hot coals, the cracking of dry wood, the creaking of frozen snow, or of leather, etc.; they may be associated with the amphoric echo, and metallic tinkling. Hence râles vary much in character; but, with very few exceptions, they indicate the presence of liquids in the bronchial tubes, or in cavities.

Most of the râles take the character of sounds produced by the breaking of bubbles in a liquid; some, however, resemble the creaking of leather, etc.; the first sort have been called moist, the latter dry râles; but no distinctive line can be drawn between them; they pass gradually one into the other. It may be asked, if we are able, by the character of the râle, to ascertain the nature and quantity of the fluid which occasions it, whether the fluid is present in the air-cells, in the larger or smaller bronchial tubes, or in cavities, and what is the condition of the lung parenchyma? These questions cannot be answered until all the varieties of sounds which these râles present, and their causes, are determined.

The sounds caused by the breaking of bubbles in a fluid, vary with the size of the bubbles; moist râles are formed by large, small, or very fine bubblings. In the creaking of leather and of snow, and in the cracking of wood, the intervals of the sounds vary in duration; and thus the dry râles have been called large, small, fine-bubbling râles, to indicate the extent of the intervals between the cracklings which form them.

Râles, whether dry or moist, and whatever the size of the bubbles producing them, may be loud or weak, clear or dull, scanty or abundant; they may also vary in pitch, and have an amphoric or metallic echo.

Moist and Dry Râles.

It is probable that the moist or dry character of a râle, depends upon the consistence of the liquid in the bronchial tubes or the cavities where it occurs; solid bodies, however, can only produce râles after the manner described above; when we speak of dry râles, we mean no more than that, in all probability, the fluid which occasions them is more tenacious than that which causes moist râles.

Size of the Bubbles.

The larger bronchial tubes and cavities can alone produce large bubbles; small bubbles, however, may occur both in the smaller and the larger bronchial tubes, and in cavities; the size of the bubbles in the larger bronchial tubes and in cavities, depends upon the quantity and quality of the fluid in them, and upon the rapidity of the current of air passing through it; but although the sound of small, and even of very fine bubbles, may be heard in the larger bronchial tubes and in cavities, yet large bubbles are always intermingled with them: the râles are never equal. A fine and equal bubbling râle can take place only in the smallest bronchial tubes and in the air-cells; it indicates, therefore, the presence of some fluid—mucus, blood, or serum—in those

tubes and air-cells, and by demonstrating the entrance of air into the air-cells, it precludes the existence of every diseased condition of the respiratory organs, which would render the entrance of air into the air-cells impossible. It has, in relation to the lung parenchyma, the same signification as vesicular breathing.

Amount of Râles Present.

This depends upon the quantity of fluid, and whether it be present in one or in several bronchial tubes, and upon the force of the respiration. If the râles are scanty, consisting of single bubblings, and the vesicular or bronchial respiratory murmur at the same time audible, then we may be certain that little fluid is present in the air-passages, provided cavities, the fluid contents of which are not disturbed by the stream of air, do not exist in the lungs. Abundant râles, unaccompanied by a respiratory murmur, or heard in combination with an indeterminate respiratory murmur, indicate, for the most part, that the bronchial tubes are extensively filled with mucus, blood, serum, etc. We may observe here, that râles may be heard either both during inspiration and expiration, or during inspiration or expiration only. Such difference is quite accidental; the fine equal bubbling râle is the only râle audible for any length of time during inspiration only, and continuing after the patient coughs.

Strength of the Râles.

Râles are at times so loud that they may be heard

passing through the mouth of the patient, and also through the walls of the thorax, without the ear or stethoscope being brought in contact with them; at other times they are so feeble, that as careful attention is required for their observation as for that of a weak respiratory murmur. The difference in the strength of râles, depends chiefly upon the extent and rapidity of the respiratory movements.

The death-rattle may be classed among the loudest of râles; it is audible through the mouth, and is produced for the most part in the larynx, the trachea, and the two bronchial trunks; but râles occur at the same time in the bronchial tubes. Râles arising in a superficial pulmonary cavity, may be sometimes heard through the walls of the thorax, without the ear being brought in contact with it, and this, too, when the respiration is neither rapid nor strong: in such cases, the râles are also generally heard through the mouth of the patient, although not produced in the larynx and trachea. Loud râles, arising in the larynx or the trachea, may be heard over the whole thorax, and so preclude the observation of any other auscultatory sign which the respiration might offer. Auscultation of the heart and arterial trunks within the thorax, may also be rendered impossible by the presence of râles.

Clearness or Distinctness of Râles.

It is often very difficult to determine the exact spot, where râles which are heard over the thorax arise; thoracic sounds pass through other media besides the air, and they do not always travel in a

straight direction; we must therefore endeavour to judge of their points of origin by considering their clearness and distinctness, whenever this is possible.

A remote râle is not so clear as a râle arising in the neighbourhood of the spot where it is observed; but a loud and distant râle may be clearer than a near and feeble one; again, râles arising in the larynx, the trachea, or either of the bronchi, may consonate within the thorax, just as the voice or the respiratory murmur consonates in the diseased states of the lung-tissue already referred to, and thus become distinctly audible throughout the thorax.

Fine, equal bubbling râles, if clear, must of necessity arise immediately beneath the spot where they are observed; consequently the air-cells at the part must be permeable to air, and the lung contain no cavities, or, at least, none of any magnitude.

Unequal or large bubbling râles may arise in cavities which lie near the surface of the lung, or, when dyspnœa is present, they may proceed from distant cavities, or enlarged bronchial tubes, or they may be consonating sounds produced in the trachea, even though the respiration be feeble.

It is not possible to define the distance from the thoracic walls at which a dull râle arises; it may proceed from the larynx, the trachea, the bronchial tubes, the air-cells, or cavities.

Pitch of the Râles.

I determine the pitch of a râle in the same way as that of a respiratory murmur; that is, by ascertaining the vowel which is used when we attempt to

imitate either it or an equal pitched respiratory murmur by the mouth.

The pitch of a râle generally corresponds to that of the respiratory murmur, which is either replaced by or accompanies it; the laryngeal and tracheal râles and respiratory murmurs are, as a rule, more acute than the vesicular râles and murmur. But there are exceptions to this in the case of the respiratory murmurs, and still greater in the case of the râles, in consequence of their pitch being so much affected by the quality of the fluid which produces them. However high or low-pitched a bronchial râle may be, it always loses by transmission, and more or less in proportion to its original feebleness, and to the remoteness of its source from the walls of the thorax; exception being made of the case in which its intensity is increased by consonance.

Acute râles in the larger bronchial tubes, when consonant, appear acute also over the thorax; did they reach the walls by mere conduction, they would become deeper. The larger or unequal bubbling râles, therefore, are only heard as acute sounds over the thorax when the conditions necessary for consonance are present, or when they arise in cavities near the surface of the lung; in this last case, however, as a general rule, acute râles are not heard unless the walls of the cavity reflect the sounds.

The acute large, or unequal bubbling thoracic râles, indicate the same condition of the lung-tissue, as bronchophony and bronchial respiration; it is, for the most part, a sign of hepatization, or tubercular infiltration of the lung, being more frequently pre-

sent in these affections than in pleuritic effusion; but for greater accuracy of diagnosis, it is always well to take into account the sounds elicited by percussion, and the general symptoms referred to in the remarks on bronchophony.

A deep and dull râle indicates the presence of mucus, blood, serum, etc., in the bronchial tubes, or in cavities, but affords no information respecting the condition of the lung-tissue. A deep, clear, large-bubbling râle, reaches the surface from distant parts by consonance, or it arises immediately beneath the surface of the lung, either in superficial cavities, or in enlarged bronchial tubes; by the aid of percussion, we may generally decide whether the râle is consonating or not.

LAENNEC'S DIVISION OF THE RÂLES.

Laennec included, under the denomination of râles, the sonorous and sibilant sounds. He proposes five divisions:—

1. Moist crepitating râle, or crepitation—"le râle crépitant humide, ou crépitation."

2. Mucous râle, or gurgling—"le râle muqueux, ou gargouillement."

3. Dry sonorous râle, or snoring—"le râle sec sonore, ou ronflement."

4. Dry sibilant, or whistling râle—"le râle sibilant sec, ou sifflement."

5. Dry crepitant râle with large bubbles, or crackling—"le râle crépitant sec à grosses bulles, ou craquement."

Under the term râles, I myself include only such

sounds as resemble the breaking of bubbles in water, or the crackling (*Prasseln*) as of burning wood, and shall therefore speak separately of Laennec's dry sonorous, and dry sibilant râles.

Laennec's Moist Crepitating Râle.

This râle, according to Laennec, is one of the most important of auscultatory signs, and I shall therefore describe it in his own words. "The moist crepitating râle evidently arises in the pulmonary tissue; it may be likened to the sound produced by gently heating salt in a pan or by the inflation of a dry bladder, or, still better, to the sound produced by the pressure between the fingers of a healthy lung distended by air, but it is somewhat louder than this; and, in addition, it has a well-marked character of fluidity. On hearing it, we at once recognize the presence in the air-cells of a fluid of the consistency of water, by which the entrance of the air into them is not prevented; the bubbles forming the râle appear extremely small. This râle, which is one of the most important, is readily recognized; and any one who has once heard it, can scarcely fail to know it again; it is a sign pathognomonic of pneumonia in its first stage, and ceases to be heard when the lung becomes hepatized, re-appearing, however, during the resolution of the hepatization; it may be also observed in pulmonary œdema, and occasionally in hæmoptysis; but in the two last cases the bubbles formed by the displacement of the air, seem somewhat larger and more moist than those producing the crepitating râle of pneumonia; to this variety of

râle I have given the name of sub-crepitant râle—*râle sous-crépitant.*"

Attempts have since been made to distinguish between the crepitating and sub-crepitating râles. Dance considered that they differed in this: that the crepitating râle was audible only during inspiration, and did not disappear after expectoration; the sub-crepitating râle being heard during both inspiration and expiration, and disappearing after expectoration.

It is quite true, that the crepitating râle of Laennec is occasionally observed during inspiration only, and is not removed by expectoration; but this does not characterize it as a sign pathognomonic of pneumonia. Andral, Cruveilhier, Chomel, etc., have brought forward numerous facts, which prove that Laennec's crepitating râle cannot be regarded as such a sign; and that, in fact, no distinctive line can be drawn between crepitating, sub-crepitating, and mucous râles. Laennec's opinion, however, of the great value, as a sign, of the crepitating râle, still prevails; and most writers are careful to make a marked distinction between this and every other râle.

For myself, I consider Laennec's crepitation, viz., the fine, equal-bubbling râle, denotes the presence of fluid in the finer bronchial tubes and in the air-cells, and the entrance of air into the latter; but that it does not inform us by what particular disease the fluid is produced; this must be learnt from other phenomena. Moreover, I have not only not found this crepitation constantly present in pneumonia, but, if we are to follow Laennec's description of it very closely, I must say that I have not often observed it.

Laennec's Mucous Râle.

Under this title are comprised the mucous râle proper, *râle muqueux*, or as Andral calls it, *râle bronchique humide*, and the cavernous râle—*râle caverneux*. Laennec distinguishes the mucous râle from the crepitating, by the greater and unequal size of the bubbles which compose it; and the cavernous from the mucous râle by the bubbles of the former being more abundant and louder than those of the latter; and by the circumstance of its being heard in a circumscribed space, where also the cavernous cough and respiration, as well as pectoriloquy, are generally present.

It will be seen at once, from this description of the cavernous râle, that it is in no respect a more accurate sign of the existence of a cavity than pectoriloquy, or cavernous respiration. The size and abundance of the bubbles depend upon the quantity and the quality of the fluid in the bronchial tubes, or in the cavities, and upon the force of the respiration. The limitation of the râle to a confined space is a very uncertain sign. If cavities occupied the whole of one lung, how could we recognize the cavernous character of the râle as described by Laennec? and how are those abundant râles which sometimes arise in large, single, and superficial bronchial tubes, to be distinguished from Laennec's cavernous râles?

I am convinced that no difference can be recognized between bronchial and cavernous râles, except in cases where the latter are associated with amphoric echo, or metallic tinkling. Cavernous, just as bronchial râles, are produced by bubbles, large and small,

moist and dry, abundant and scanty, clear and dull, high and deep toned, and may consonate, like the bronchial. Cavities in the lungs, and bronchial tubes equally, may remain for a long time filled with fluid, without producing any râles; râles only arise in cavities, when these can expand and contract during respiration, and when the passage of air into and out of them is not wholly obstructed.

Laennec's Dry Crepitating Râle with Large Bubbles—*Craquement.**

This râle, according to Laennec, resembles the sound produced by inflating a pig's bladder, and is a sign pathognomonic of vesicular and interlobular emphysema of the lungs; it is observed only in those cases where the air-cells of a portion of the lung are considerably enlarged, to the size of a pea, perhaps, or a bean, and communicate with the bronchial tubes: in no other case of pulmonary emphysema is it present. It may arise, moreover, in enlarged and sacculated bronchial tubes, and in cavities of the lungs which have membranous walls, and communicate with the bronchial tubes through moderate-sized openings.

Laennec's craquement has been attributed to a tearing up of the pulmonary tissue; but I do not

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* No one, since Laennec, appears to have heard this râle, and it is for the most part excluded from the list of auscultatory phenomena. I cannot agree with the general opinion on this point, for I consider that such a râle really exists. However, he who has not heard it, or has not been able to discriminate it, has not lost much.

consider that we have any knowledge whatever of the sound produced by rupture of the air-cells. My own belief is, that the craquement is caused by distension, during inspiration, of the walls of the air-cells, the bronchial tubes, and cavities, when these walls have lost their contractility, and simply collapse during expiration.

Such collapse, or, more properly speaking, compression of the air-cells, enlarged bronchial tubes, and cavities, is only possible when the still contractile portion of the lung—on account of some obstruction of its bronchial tubes, or of the large extent of tissue rendered non-contractile in consequence of the adhesion of the lung to the costal pleura, etc.—is insufficient, when inflated by inspiration, to distend the cavity of the thorax; the non-contractile portion must therefore become altered in volume during the respiratory movements.

FOURNET'S DIVISION OF THE RÂLES.*

His chief distinctions are drawn from the seat of origin of the râles:—

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* Barth and Roger follow Laennec's division of the râles; they, however, call the mucous râle—*râle sous-crépitant.* According to Professor Chomel, certain special characters belong to the cavernous râle, by which an observer may decide whether it is produced in a pulmonary cavity, or in a circumscribed pleuritic cavity, communicating with bronchial tubes. "A cavernous râle is limited and fixed, and is generally situated in the upper part of the thorax; its force diminishes in every direction, as we follow it from the centre of the cavity; but in the case of pleuritic effusions, a large bubbling gurgling is produced towards the base of the lung, and is propagated upwards,

1. Intra-vesicular râles.
2. Extra-vesicular râles.
3. Bronchial râles.
4. Tracheal râles.
5. Laryngeal râles.
6. Bucco-pharyngeal râles.

Of the vesicular râles—*râles intra-vésiculaires*—he offers the following varieties.

1. Râle humide à bulles continues de la congestion sanguine.
2. Râle sous-crépitant de l'œdème pulmonaire.
3. Râle sous-crépitant du catarre pulmonaire aigu capillaire.
4. Râle sous-crépitant, ou crépitant de retour de la pneumonie.
5. Râle crépitant primitif de la pneumonie.

These râles are classed according to their degree of dryness or moisture; number one representing the moistest, and number five the driest variety of vesicular râles.

Under the term *extra-vesicular râles* are included:

1. Le râle ou bruit de froissement pulmonaire.
2. Le râle de craquement sec.
3. Le râle de craquement humide.

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according to the direction which the bubbles take, in passing through the fluid."

One cannot help asking, What becomes of the air-bubbles which pass through the fluid during each inspiration?

I shall refer to this hypothesis of the rising of air-bubbles, when I speak of metallic tinkling. In my opinion, Chomel has interpreted his facts wrongly, no such distinctions as these described by him really existing.

4. Le râle cavernuleux ou muqueux à timbre clair.

5. Le râle caverneux humide ou de gargouillement, et le râle caverneux sec.

The two first râles arise external to the air-cells, and are produced by friction of the lung-tissue against hardened parts; the two last arise in cavities. The third variety is heard just about the period when the hardened parts begin to soften down.

The bubbling râles in the bronchial tubes are:

1. Le râle de gargouillement dans les cas de dilatation considérable des bronches.

2. Le râle muqueux à grosses bulles.

3. Le râle muqueux à bulles moyennes.

4. Le râle muqueux à pétites bulles.

The fine, bubbling râles of the bronchial tubes are heard both during inspiration and expiration; the vesicular râles during inspiration only.

What is meant by tracheal and laryngeal râles, is sufficiently indicated by their names.

The bucco-pharyngeal râle implies a fine crepitation, which is heard when the ear is brought near to the mouth of a patient whose air-passages contain some kind of fluid.

The mucous râle with large bubbles, Fournet observed during inspiration, in two cases of purulent infiltration of the lung; and he makes the remark, that further experience may perhaps prove such a râle to be a sign of the passage of pneumonia from its second to its third stage.

I have no remark to make upon the vesicular râle of Fournet, further than what I have already said when speaking of Laennec's crepitating râle. I

do not deny that the varieties of râles described by Fournet really exist, I believe that many more might be added to them; but I affirm, and my opinion is that of many other auscultators, that there is no one distinct râle peculiar to congestion of the lungs with blood, or to pneumonia, or to œdema, or to catarrh, etc.: the classification of râles according to the nature or the stages of disease, is, in my opinion, erroneous.

Of the bruit de froissement pulmonaire, Fournet says: "The general character of this bruit is, that it produces in the ear a peculiar sensation of rubbing, by which it may be always readily recognized. The observer fancies that he both sees and hears the pulmonary tissue forcibly struggling against some impediment to its expansion."

"The bruit appears in different forms and degrees. When most perfect, it resembles the bruit de cuir neuf, and differs from the new leather friction-sound of pericarditis solely in being of more acute timbre; when less perfectly marked, it appears as a plaintive, moaning sound, and presents various intonations, according to the state of the patient and the force and rapidity of the respiration; lastly, in its third and most feeble form, which is the least frequently met with, it reminds the observer simply of the gentle, rapid, and dry sound which is obtained by blowing upon very fine and dry paper."

Several varieties of murmurs are included under the denomination of froissement, all of which have this character in common, that they give to the person observing them the impression that he can see

the lung-tissue struggling, with force and noise, against some obstacle to its distension. I have never yet met with such a struggling sound, and shall abstain from offering any opinion upon it until its existence shall have been clearly demonstrated. According to Fournet, it is scarcely ever observed, except in the first stage of phthisis, particularly during the acute development of miliary tubercles. He looks upon it as an important sign of phthisis in its first period. I shall mention the phenomena which I have myself observed in cases of miliary tubercular disease, in the chapter on tuberculosis.

Craquement is a dry râle, and indicates the presence of fluid, probably of a tenacious character, in some of the bronchial tubes, or in a cavity. This dry alternates with moist râles, etc., not only in tuberculosis, but in all conditions of the lungs where there is fluid in the bronchial tubes. It is confined to a circumscribed space, and this is one of its most striking characters, especially in phthisis; its signification, when present in this disease, is the same as that of moist râles, and I therefore see no reason why it should be treated as a special symptom, and separated from other râles.

The râle of small cavities—*râle cavernuleux à timbre clair*—is that to which I give the name of consonating; although it is very frequently observed in certain stages of phthisis, as pointed out by Hirtz and Fournet, it has not the signification attached to it by these observers. It is not characteristic of small cavities, but of tubercular, or pneumonic infiltration of the lung-tissue.

Respecting the moist and dry cavernous râles, as well as the blowing râle of the bronchial tubes, I must refer the reader to the chapter on Laennec's mucous râle. I consider that the distinction which has been drawn between the fine bubbling bronchial râle, and the vesicular râle, viz., that the first is heard during both inspiration and expiration, and the latter only during inspiration, is arbitrary, and not supported either by observation or by theory.

In conclusion, I must observe that I am convinced there are no râles which specially indicate the passage of pneumonia from its second into its third stage; for this particular period of the disease may be attended by a great variety of râles, or it may terminate without giving rise to any râles whatever.

THE AUTHOR'S DIVISION OF RÂLES.

I divide the râles as I do the voice and the respiration, so far only as the division has a practical value. I class them as follows:

1. Vesicular râle.
2. Consonating râle.
3. Dry, crepitating râle, with large bubbles, or craquement; this râle has been already described.
4. Indeterminate râles.
5. Râles accompanied by amphoric resonance and metallic tinkling; of these I shall say more hereafter.

Vesicular Râle.

By this term, I understand (in accordance with Andral and Laennec) the râle which takes its origin in the finer bronchial tubes, and in the air-cells: we

know that the râle arises there, from the fact of its bubbles being small, and of equal size; it indicates the presence of mucus, blood, or serum, etc., in the finer bronchial tubes and in the air-cells, and the entrance of air into the latter; precluding the existence of any of those abnormal conditions which prevent its entrance. The râle, unless it is very distinctly heard, does not enable us to draw any conclusions as to the condition of the portion of the lung nearest to the surface of the thorax.

Consonating Râlé.

This râle is clear and high, is formed by unequal bubbles, and accompanied by resonance, which has neither an amphoric nor a metallic character. A high and clear râle (as already shown) cannot be produced within the thorax, unless the conditions necessary for the production of consonance are present. Hence the consonating râle is of the same significance as bronchial breathing and bronchophony; generally speaking, it indicates the presence either of pneumonia, or of tubercular infiltration, being seldom observed in pleuritic effusion.

Indeterminate Râles.

Under this head, I comprise all those râles which are neither vesicular nor consonating, and are not accompanied by amphoric resonance or metallic tinkling; they offer no special indications respecting the condition of the lung-tissue. What may be learnt by their aid, of the quantity and quality of the fluids contained in the air-passages, as well as

concerning the situation of such fluids, has been already considered, when we spoke of the different varieties of râles.

Sonorous, Whistling, and Hissing Sounds.

Laennec, as we have already observed, included these sounds among the râles. A great variety of sounds are produced by the passage of the air through the air-tubes, when certain parts of them have become narrowed from any cause. These sounds are generally known to us under the names of snoring, whistling, hissing, etc. (*Schnurren, Pfeifen, Zischen*, etc.). Their intensity, and the force with which they strike upon the ear, enable us to judge, but only approximatively, of the size of the bronchial tubes in which they are produced. The sonorous sounds are most commonly produced in the larger bronchial tubes; the whistling in bronchial tubes of a less diameter, and the hissing in the finest portions of the air-passages; but there are numerous exceptions to these rules. The proximity of the point of origin of a sonorous, or whistling sound, cannot be judged of by the distinctness with which it is heard. These sounds may be frequently observed of equal strength over a considerable portion, or even over the whole of the thorax: and the observer may sometimes hear them without bringing his ear in contact with the thorax, and even at a considerable distance from it.

Snoring, whistling, and hissing sounds, may arise, when the lung tissue is normal, or under any of its abnormal states; they therefore afford no information as to its condition, except when they are con-

sonant. They consonate, in fact, just like the voice, the respiration, and the râles. Any one well acquainted with the bronchial breathing, will have little difficulty in deciding when these sounds are consonant, and when they are not so; when consonant, they are accompanied by a resonance resembling that of the bronchial voice; and offer the same indications as to the condition of the lung-tissue, as bronchial breathing, bronchophony, etc. The sonorous, whistling, and perhaps also the hissing sounds, may also be attended by amphoric echo and metallic tinkling. The sonorous sound sometimes passes into dry crepitation.

III. AMPHORIC ECHO AND METALLIC TINKLING. (Bourdonnement amphorique et tintement métallique).

These phenomena may be imitated by a person speaking and directing his voice into a jug; when he does so, a peculiar humming is heard, in addition to the voice; this humming is Laennec's amphoric buzzing. The strength of the voice is at the same time generally increased, and very remarkably so, when of a certain pitch. The humming which accompanies the voice is not always of the same pitch as the voice; it may retain its pitch, though that of the voice be altered.

Besides this humming, there is also occasionally heard a metallic after-tone (*Nachklang*), resembling the flageolet-tone of a guitar string. This tone, when it accompanies the voice, exactly represents Laennec's metallic tinkling. It may be often heard, as a kind of metallic echo, in rooms, particularly if arched,

when a person is speaking rather loud, and at a particular pitch of the voice. Any one may convince himself, by the production of the metallic echo in a room, and by directing his voice into a vessel, that amphoric echo and metallic tinkling are phenomena which arise under similar conditions, and that metallic tinkling bears the same relation to amphoric resonance, as does a high to a deep flageolet tone of a guitar-string.

Amphoric echo and metallic tinkling cannot be produced in narrow tubes; and therefore do not arise within the thorax unless there is some large cavity present, the walls of which are capable of reflecting sound. This statement is fully corroborated by clinical observation, these phenomena being only met with in cases of pneumothorax, and where large pulmonary cavities exist.

Laennec believed that these sounds were not produced, unless both fluid and air were present in the cavity; I do not myself consider that the presence of fluid is necessary. The two phenomena are equally well produced in a pitcher, whether it contain fluid or is perfectly empty; and assuredly the presence of fluid is not requisite to excite the metallic echo in a room. If a person speak into a stethoscope placed on a stomach filled with air, both metallic tinkling and amphoric echo will be heard sounding within the stomach, and this, whether the stomach be partly filled with, or contain not a drop of fluid.

Laennec believed, moreover, that these phenomena could not be produced by the voice, unless a communication existed between the pulmonary or pleu-

ritic cavity and some bronchial tube; but it very rarely happens that in pneumothorax the communication between the air in the pleura and that in the bronchial tubes remains free; and yet we seldom meet with a case of pneumothorax in which these sounds are not present. In the experiment with the stomach just referred to, metallic tinkling is produced, though no communication exists between the air in the stomach and the air in the stethoscope: from this experiment also we may learn how the air in the pleura is made to vibrate by the voice in the larynx; if, for instance, the voice consonates in a bronchial tube, which is separated from the air in the pleural cavity merely by a thin layer of pulmonary substance, its sound will pass into the air of the cavity with sufficient force to excite therein consonant vibrations.

Pulmonary cavities, if tolerably large, always communicate with the bronchial tubes. I am not able to state what is the smallest size of a pulmonary or a pleural cavity, that admits of the production within it of amphoric echo and metallic tinkling.*

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* In a treatise by Kolisko (*Öst. Jahrbuch, Okt.* 1844) on amphoric echo and metallic tinkling heard in cavities within the thorax, my opinions — viz. that these sounds may arise when there is no communication existing between the air in the pleura and the air in the trachea, and that they may be excited by sounds originally produced in a neighbouring bronchial tube, or by sounds which have been strengthened by consonance—are confirmed; and a case is related, where a single tubercular cavity, of the size of a pigeon's egg, and lined with false membrane, gave rise to a distinct metallic tinkling. Kolisko's explanation of the phenomena may be seen in his treatise.

If amphoric echo and metallic tinkling are heard during respiration in pneumothorax,—where, as before mentioned, the air in the pleura rarely communicates with the air in the bronchial tubes,—the laryngeal or the tracheal respiratory murmur must have become consonant in a bronchial tube, which is separated from the pleural cavity by merely a thin layer of lung-substance.

Amphoric echo and metallic tinkling are produced in pulmonary cavities by the drawing in and forcing out of the air; the transition of the amphoric echo into the metallic tinkling is best observed during respiration. The respiratory murmur may in one case resemble a deep humming, like that caused by blowing into a jug; in another, or even in the same case, at some other period, it may resemble the deep whistling which is produced by drawing in or forcing out air from the mouth when its cavity is enlarged and the opening of the lips narrowed; this sound being heard alone or combined with the humming. The deep whistling, which evidently represents a tone (*Klang*), may be replaced by a more acute and also by the essentially metallic tone, viz., that which resembles the flageolet-tone of a guitar-string, and which may continue during the entire act of inspiration and expiration.

Metallic tinkling is more frequently excited in pneumothorax, and in large cavities, by râles, than by the voice, or the respiratory murmur; the râles can take this metallic character, although no communication exists between the pneumothorax and the bronchial tubes, and although no fluid be present with the air in the pulmonary or pleural cavity.

Dr. Dance has offered the following explanation of the mode of origin of the metallic tinkling (as may be seen in Raciborsky's manual of auscultation and percussion): "When the level of the fluid contained in the pleural cavity is higher than the opening through which the air enters it, the air, at each inspiration, is drawn into the cavity, and, in consequence of its specific gravity, rises in bubbles to the surface of the fluid, where the bubbles break, and so produce metallic tinkling."

In this explanation, we are not told what becomes of the air which rises above the level of the fluid; it is evident that, like the air in a healthy lung, it must be either taken up, other gaseous bodies being separated in its place, or it must be very gradually absorbed, or must remain unabsorbed. Under any of these conditions, it is difficult to understand how the metallic tinkling can continue audible after a few inspirations, or even after one single inspiration: for, in fact, the cavity takes in at each inspiration as much air as it can contain; and if the level of the fluid be higher than the opening into it, the air cannot be forced out of it during expiration. The cavity, during expiration, remains either completely distended, and consequently cannot admit more air at a subsequent inspiration; or it is compressed, and then a portion of its contents must be forced into the mouth of the opening leading into it, from whence it is drawn back again into the cavity at the next inspiration. According to this explanation of Dr. Dance, the phenomenon of metallic tinkling should be very rarely observed, and only at considerable intervals.

and more particularly during inspiration after coughing; how it should ever arise, during expiration, is perfectly incomprehensible.

Dr. Beau, who admits Dr. Dance's theory, explains it thus: "In most cases", he says, "the cavity is surrounded by condensed parenchyma, and its capacity does not diminish during expiration; consequently the air which is forced out of the other parts of the lungs, either during expiration, or by coughing, speaking, or expectorating, presses from the trachea into the gaping bronchial tubes, and then acts just as though it had been inspired."

Dr. Beau does not tell us whether air, under these circumstances, enters into the cavity during inspiration, but we are bound to believe that it does, inasmuch as metallic tinkling is heard during inspiration: it would appear, therefore, according to his explanation, that cavities surrounded by condensed tissue, receive air both during inspiration and expiration, and yet cannot force any of it out again!

My own opinion is, that metallic tinkling—in addition to its being heard as a resonance of the voice, of the breathing, and of whistling sounds—may present itself in large cavities, as the resonance of a râle, situated in a distant, but communicating bronchial tube, or as the resonance of a râle which has its origin at the mouth of the opening into the cavity, or when many cavities communicate together, at the opening of communication between them—where, in fact, the air may pass in and out during respiration, without being obstructed by fluid—or as the resonance of a râle produced in cavities, through violent concussion of their fluid contents, by coughing, etc.

The metallic tinkling of pneumothorax is produced in a similar way; but as the air contained in the pleura very rarely communicates with that in the bronchial tubes, the metallic tinkling of pneumothorax is more commonly produced as a loud, or as a consonating râle, arising in a large and neighbouring bronchial tube, or by succussion of the fluid in the thorax, during coughing, etc.*

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* If the pulmonary pleura be destroyed or torn at a part of its surface, where it is not adherent to the thoracic walls, and an opening for the entrance of air from the neighbouring parts of the lungs into the pleura thereby established, it will be found that the air enters into the pleural cavity, both during inspiration and expiration, so long as the contractile power of the lung remains; when its contractility ceases, air enters into the cavity during inspiration only, and continues to do so until the cavity is capable of no further distension. No air can escape from the cavity during expiration, for the opening at the surface of the lung, through which the air enters into the cavity during inspiration, is closed by the pressure of the air within the cavity upon the surface of the lung during expiration: an exception must be made of the case where a canal, with resistant walls, leads from the cavity into the trachea—a case, however, which is rarely ever met with.

In pneumothorax produced by the bursting of a pulmonary cavity, or abscess into the pleura, the communication between the air in the cavity and the air in the trachea is completely destroyed after a few inspirations; however small the opening may be, the pleura becomes very rapidly filled with air.

The communication is, in some rare cases, restored by ulceration of the compressed lung, when the ulceration has perforated or laid bare an incompressible bronchial tube, or even the trachea, or when a single-mouthed fistulous opening into a large bronchial tube, or into the trachea, has been formed in a lung consolidated by infiltration.

A drop of fluid, or a solid body falling accidentally to the bottom of a pleural cavity filled with air, would undoubtedly give rise to metallic tinkling; but such a cause of the phenomenon must be exceedingly rare.

When metallic tinkling presents itself as the resonance of a whistling sound, it resembles the most beautiful tone of a guitar-string, when a bow is drawn across it.

IV. THE SIMULTANEOUS EXISTENCE OF THE RESPIRATORY MURMURS, RÂLES, AND SONOROUS SOUNDS.

It often happens that several distinct sounds are heard simultaneously, during respiration; thus, râles, and whistling and sonorous sounds, and the proper respiratory murmur, may be all recognized by the ear at the same time. But these murmurs, râles, etc. are not met with mixed together promiscuously.

The non-consonating râles, and the non-consonating sonorous, whistling, and hissing sounds may be heard in company with the vesicular respiratory murmur, provided they are neither so loud nor so extensively distributed through the lungs, as to obscure the murmur. The vesicular murmur is never associated with metallic tinkling, and amphoric resonance. Bronchial respiration and consonating râles, are sometimes heard in company with it, when the deeper parts of the lung are in the condition necessary for the production of consonance, whilst its surface contains air; but such an abnormal state of the lungs seldom occurs, except in those particular cases of pneumonia where the inflammation travels

from one part of the organ to another, attacking each in succession. In cases of violent dyspnœa, however, where the breathing is loud, it not unfrequently happens that the vesicular and the bronchial murmurs are heard together, particularly over the dorsal region, independently of any consonance.

The non-consonating respiratory murmurs of the larger bronchial trunks, which I place among the indeterminate murmurs, are more frequently than bronchial breathing heard in company with the vesicular murmur. Consonating and non-consonating râles, sonorous, whistling, and hissing sounds, of every variety, as well as the indeterminate respiratory murmurs may be associated with the bronchial respiration.

Bronchial respiration may also be conjoined with amphoric resonance and metallic tinkling, without being masked by these sounds.

The indeterminate respiratory murmurs may be attended by râles, and sonorous, whistling, and hissing sounds of every kind; and the same is true of amphoric resonance and metallic tinkling.

The simultaneous existence of several murmurs, renders the accurate discrimination of any particular one very difficult; it is only after long experience that the ear is able to distinguish between such sounds, so as to isolate any one of them from the rest, and thus examine each in turn.

The concurrence of two murmurs, in which the distinction is not readily determined even when they arise separately, occasions the utmost difficulty in diagnosis; the combination of vesicular and bronchial

respiration is an instance: the two sounds blend together into one, and the nicest observation is barely sufficient to discriminate the one from the other.

If, in such a case, the expiratory murmur gives no sure indication respecting the condition of the lung, that is, if it be not distinctly bronchial, the respiratory murmur must be considered as indeterminate, and the diagnosis established by some other means. A murmur resembling that produced by the combination of the vesicular and the bronchial respiration, may arise through other causes besides those which occasion the simultaneous occurrence of the vesicular and bronchial respiration.

It is difficult to determine the clearness and pitch of a râle, when it is associated with a whistling or a hissing sound; nor can we draw conclusions from the clearness and pitch of whistling sounds in the same manner as we can from those of the râles under similar circumstances. If we wish to ascertain the clearness and pitch of a râle which is combined with whistling or hissing sounds, we must detach it entirely from them; and if this cannot be done, the râle must be set down as incapable of yielding information respecting the condition of the lung. If the observer attributes the pitch of the whistling sound to the râle, he will mistake many murmurs for consonating, which are, in reality, not so.

V. AUSCULTATION OF THE COUGH.

The cough does not afford us any signs different from those which have been already described; but it may render them all more distinct, as, for instance,

by removing the accumulation of fluids, etc. which frequently takes place in the bronchial tubes, and obstructs the passage of the sounds. Coughing produces in the larynx its known peculiar sound, or a lesser degree of the sound which is peculiar to expiration, or it excites different kinds of râles. In the bronchial tubes, or in pulmonary cavities, the cough gives rise either to the ordinary expiratory murmur alone, or to this murmur and, in addition, to râles, and whistling and sonorous sounds.

The sound peculiar to coughing, and the other sounds excited by it in the larynx and the air-passages, are heard over the thorax in varying degrees of strength and clearness, according to the laws of consonance and conduction of sound which have been already referred to. Thus the coughing may be heard as a bronchophonic sound, or as an indistinct humming, and the respiratory murmur caused by the cough, as a bronchial or indeterminate respiratory murmur, and the râles as consonating or indeterminate. In cases of pneumothorax, and where large pulmonary cavities are present, the cough may excite metallic tinkling and amphoric resonance. After coughing, the patient generally inspires more deeply than ordinarily, and thereby renders the inspiratory murmur more distinct.

Laennec made a distinction between the tubular and cavernous cough, and the dull cough-sound which is heard in the normal condition of the lungs. On this point, I have only to repeat what has been already said respecting his division of the thoracic voice.

VI. FRICTION SOUNDS PRODUCED BY THE ROUGHENED SURFACES OF THE PLEURA DURING THE RESPIRATORY MOVEMENTS.

The contraction of the diaphragm causes the cavity of the thorax to enlarge downwards during inspiration, the lung, by the pressure of the air, being made to occupy the augmented space, and therefore to descend a little. When the contraction of the diaphragm ceases, the lung contracts and ascends, regaining its former volume. The relaxed diaphragm follows the movement of the lungs upwards, the atmospheric pressure preventing the existence of any vacuum in the thorax, or of any interval between the diaphragm and the lung—provided no gaseous or fluid matters intervene. The upward movement of the diaphragm is frequently promoted by the elasticity of the abdominal viscera, and contraction of the abdominal muscles.

This ascent and descent of the lung occasions a rubbing between the pleural surfaces, which is increased by the upward movement of the fore part of the thorax during inspiration, and its downward movement during expiration, the lung moving in the contrary direction.

Diminished distensibility in one part of a lung causes increased movements in other parts of it, and consequently greater friction of the pleural surfaces. If, for example, a portion of lung does not expand, the parts around are pressed in towards it at each inspiration, and fill the space which normally would have been occupied by the non-expanding portion of

lung; at each expiration, the parts return back to their former position.

This contact of its surfaces produces no friction-sound, so long as the pleura remains smooth and moist; but the sound manifests itself whenever, through any cause, the surfaces have become roughened; it generally accompanies both inspiration and expiration, being at one time most distinct during inspiration, at another during expiration; it may be heard during inspiration only, or the reverse. It resembles the creaking of leather, appears at intervals, and is only distinguishable from a dry râle by producing a feeling of friction and momentary contact (*Anstreifen*). In most cases, it is recognizable by the finger as well as by the ear, and the patient generally experiences the sensation of something rubbing within his thorax. Laennec has given to this friction-sound the name of *frottement ascendant et descendant;* it is, in fact, almost always produced by the vertical motion of the lungs, but it may be occasionally produced by a horizontal movement, when any such impediment to the distension of the lungs exists as to cause a portion of these organs to take the horizontal direction during inspiration.

Laennec imagined that the friction-sound was in most cases produced by superficial interlobular emphysema, *i.e.*, by the air vesicles immediately beneath the pleura. He also supposed that it would manifest itself when any cartilaginous, bony, tubercular, or schirrous tumours projected above the surface of the lung.

Dr. Reynaud showed that the friction-sound was

generally caused by roughened pleural surfaces, and his views have since been fully confirmed. The sound is most commonly caused by pleurisy. It is sometimes heard at the commencement of this disease, when lymph has been deposited on the surfaces of the pleura, and the contact of the surfaces is not prevented by serous effusion; at this period of the disease, however, the lymph has not always sufficient consistency to produce the sound. It is much more frequently heard, and more distinctly marked, at a later period of the disease, when absorption of the serous effusion has taken place, and the surfaces, covered by a firm plastic exudation, have once more come in contact. In this latter case, the friction-sound continues, until either the lung has formed attachments to the thoracic walls, or the rubbing surfaces have become perfectly smooth.

Tumours of the nature above referred to,—bony, cartilaginous, tubercular, etc.—do not produce friction sounds, if their surfaces are smooth; neither does interlobular emphysema.

There is no doubt that a friction-sound may be produced by the rubbing of one lobe of a lung against another; but it would be difficult to distinguish such a sound from a dry râle. The friction sound varies both in the length of its intervals, and in its strength. Differences in its strength, which depend chiefly on the extent and the rapidity of the respiratory movements, render the sound more or less distinct. The friction-sound may be limited in extent or heard over many inches of surface.

CHAP. IV.

AUSCULTATORY PHENOMENA PRESENTED BY THE ORGANS OF CIRCULATION.

These consist chiefly of the sounds and murmurs which attend the heart's movements, and are audible in the præcordial region, and in several of the arteries. By auscultation, however, we not only hear, but we also feel the beat of the heart against the walls of the thorax, and occasionally also the pulsation of the arteries; such impulse of the heart and arteries must consequently be reckoned among auscultatory phenomena, in so far as it is made sensible to us by auscultation. Auscultation, again, takes note of the rhythm of the heart's movements.

I. THE IMPULSE OF THE HEART.

Cause of the Heart's Impulse.—By impulse of the heart is understood its beat against the walls of the thorax, which is observed nearly synchronously with the pulsation of the carotid arteries, and generally about the cartilages of the fifth or sixth true ribs on the left side.

Corrigan, Stokes, Rigeaux, and Burdach, believed that the impulse of the heart occurred during the diastole of the ventricles, not during their systole, an opinion now generally admitted to be erroneous: it does not require refutation. The same remark

may be applied to the explanation of the impulse, by a supposed lengthening of the ventricles during their systole, or by a stretching of the aortic arch.

We may also pass over Dr. Hope's views, as they have never been seriously entertained by later observers.*

Bouillaud and Filhos† explain the impulse in the following manner: "The muscular fibres of the heart have their fixed point at the tendinous rings around its base, and run from thence in a spiral direction towards its apex; now these fibres become shortened during the ventricular systole, and consequently, the apex, like the moveable end of a lever, is tilted upwards, and towards the walls of the thorax. Filhos affirms, that the impulse is produced solely by the contraction of the left ventricle, the muscular fibres of the right not being spiral, and therefore incapable of other movements than those of contraction and dilatation.

The last opinion is completely refuted by clinical observation; for, in cases of hypertrophy, with dilatation of the right ventricle, the impulse is often very strong, when the left ventricle is of its normal size, or even atrophied. I have never been able to convince myself that the apex of the heart is raised towards the thoracic walls during the ventricular systole, in consequence of the arrangement of its muscular fibres, but I am far from denying its pos-

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* Dr. James Hope, *Diseases of the Heart and Arteries.*

† *Traité clinique des Maladies du Cœur*, par J. Bouillaud. Paris, 1835.

sibility. This much however is certain, that the simple raising of the heart's apex towards the thoracic walls does not explain all the phenomena which accompany the impulse.

When the heart acts vigorously in thin individuals, we may observe, from the projection of the intercostal spaces, that it moves downwards during the systole, and returns to its former position during the diastole. The beat of the heart is sometimes felt at the scrobiculus cordis, and an elevation of the parts there, observed during each ventricular systole, which disappears during the diastole.

Dr. Gutbrod* gives the following explanation of the cause of the heart's impulse :—" It is a well-known physical law, that when a fluid escapes from a vessel, the equality of pressure produced by the fluid on the walls of the vessel is lost, for there is no pressure at the opening whence the fluid escapes : but at that part of the vessel which is opposite to the opening, the pressure is still exerted. This pressure it is which set Segner's wheel in motion, and produces the recoil of fire-arms, etc. By contraction of the ventricles, the pressure which the blood exerts upon the walls of the heart, opposite to the opening whence the stream escapes, causes a movement of the heart in a direction contrary to that of the stream of blood,

* I do not know when this theory was first brought forward by Gutbrod, but it is certain, that a precisely similar explanation of the cause of the heart's motion, and identical illustrations of it, were published by Dr. Alderson, as long ago as the year 1825, in the eighteenth volume of the *Quarterly Journal of Science, Literature, and the Arts.* (Transl.)

and by this movement the impulse of the heart against the walls of the thorax is produced. The heart is driven in a direction contrary to that of the arteries, with a force proportionate to the quantity and the velocity of the current of the blood.

Many objections have been made to this explanation. Valentin, in his *Repertorium* for 1841, made the following statement:—"When an opening is made into the apex of a frog's heart, no diminution nor other change is observed in its impulse; but some change ought to follow, on the supposition of the impulse being caused by the counter-pressure of the blood." To this I replied, that there was no evidence whatever that the impulse of the frog's heart was produced by the counter-pressure of the blood; for the frog's heart has no downward movement. Now, if the counter-pressure of the blood has no influence on the impulse of the frog's heart, the cause of this must lie, either in the slowness of its contractions, or in the small amount of its blood. To answer this objection, Valentin cut off the apex of the heart in a rabbit, and fixed a glass tube into the opening, to prevent its closure; and still he observed no change in the heart's impulse. I am far from saying that Valentin's observations are erroneous; but I am not at all inclined to pass over, without consideration, the experiments which have determined the fact of the descent of the heart, under certain circumstances, during each systole. Among the theoretical considerations by which Valentin seeks to prove the inapplicability of Dr. Gutbrod's explanation, the last conclusion is, in my opinion,

incorrect. *Vide* Valentin's *Lehrbuch der Physiologie*, B. i, p. 426.

Dr. Messerschmid makes the following remarks upon Gutbrod's theory, in Froriep's *Notizen* (Jänner. 1840, No. cclxvi, p. 29) :—" The expression, *finds* no counter-pressure, is founded on an erroneous supposition. In fire-arms, and at the mouths of the openings of Segner's wheel, there is the pressure of the atmospheric air, and at the openings of the heart, that produced by the columns of blood in the arteries; hence this explanation of the heart's impulse rests on an error, and the ordinary explanation of the movements of Segner's wheel is unsatisfactory."

Any one who will carefully examine the abovementioned physical law, will learn that the expression, " finds no counter-pressure", does not refer to the medium surrounding Segner's wheel, but that the question in debate is, concerning the pressure and counter-pressure of the fluid upon the walls of the vessel containing it.

The pressure of the columns of water on Segner's wheel is not destroyed, and consequently its motion not lost, *in vacuo*. All that we have therefore to decide is, whether the surrounding air has any, and, if any, what influence upon its movements. Dr. Messerschmid says :—" Has any one attempted to set Segner's wheel in motion by water, in a very perfect vacuum ? I doubt whether it has ever been done. But, independently of this, it is certain that the backward revolution of this machine is not caused by the one-sided pressure of the water solely, for the surrounding air has also a considerable share

in producing it. The air presses uninterruptedly against the jets of water from the openings of the horizontal tubes, and through these jets the counter-pressure is propagated inwards to those parts of the walls of the tubes which are opposite to their openings. And hence, in consequence of the well known arrangement of this machine, the counter-pressure of the external air becomes the chief cause of its backward movement."

It does not appear very clear why the counter-pressure of the air should be supposed to play a subordinate, though considerable, part in the movement at first, and afterwards become its principal cause; nor is it very intelligible, why the assumed counter-pressure should limit its influence merely to that part of the walls of the tube which is opposite to the point at which the fluid escapes. To carry the argument out logically, this counter-pressure ought to force the vertical column of water upwards, and at last cause it to flow out over the upper opening! How comes it that the air resists the stream of water, and presses it back, whilst the vertical pipe offers no resistance to the backward movement?

It is indeed surprising, how any one, on mature reflection, can believe that the resistance offered by the air to the flowing water, is capable of operating backwards through the stream of water. All that really takes place as regards the air is, that a portion of it is displaced by the water; the barometrical pressure of the air has nothing to do with the phenomenon.

Neither does the pressure of air in any way assist

in the recoil of fire-arms; here, also, there is merely a displacement of a certain quantity of air. If the air in and around the mouth of fire-arms opposed the forward movement of the expanding gases, any tendency to a backward movement that might be thus produced in them, would of necessity be compensated by the resistance of the air behind the fire-arms; for there is no reason why the air in front of the fire-arms should be displaced with greater difficulty than the air behind them. The cause of the recoil of the fire-arms depends altogether upon the pressure exercised by the expanding gases upon the closed end of the fire-arms, which pressure is not compensated by any counter-pressure of the gases at the opposite end (where the mouth is).

It is true that nothing like the expansion of gases takes place in the heart, nor is there in its conditions the slightest similarity to those which obtain in Segner's wheel; but there is, nevertheless, no reason why the recoil of the heart may not be produced on the same principle as the recoil of fire-arms, and the movements of Segner's wheel. During the ventricular systole, the blood presses upon every part of the heart's surface with a force equal to that by which it is itself compressed; the pressure of the compressed blood on that part of the heart's walls which is opposite to the arterial openings, produces a movement in a direction contrary to these openings; the compressed blood exerts no pressure there, in consequence of the walls of the cavity being deficient.

The resistance which the blood contained in the

arteries offers to the flow of blood from the heart, has nothing to do with the physical laws referred to by Dr. Gutbrod and myself; it is not because I do not think this resistance really exists, that I have not mentioned it. The movement of the heart downwards can only take place, according to the laws referred to, when all resistance is overcome, that is to say, when, for our purpose, it no longer exists. It is only the surplus of the expulsive power of the heart over the resistance of the blood in the arteries, which is taken into account in this explanation.

Professor Von Kiwisch says, in vol. ix of the *Prager Vierteljahrschrift*, p. 501:—"Still less tenable (than Gendrin's theory) is Gutbrod and Skoda's explanation of the cause of the heart's impulse; according to their view, the general pressure of the blood contained in the ventricles of the heart, is removed by the escape of the blood through the single opening, and is made to bear, in an especial manner, upon that part of the walls of the heart which lies opposite to the opening, and thus forces the heart downwards. The same physical law which moves Segner's wheel, and causes the recoil of discharged fire-arms, is the law in action here."

"The incorrectness of the above explanation is rendered manifest by the fact that, in the heart, the motor power does not cease to act even during the systole, when the influence of the blood flowing from the auricles is removed by the closure of the auriculo-ventricular valves. We must also remember that, in this comparison, an apparatus is cited, which is formed of rigid materials, and a good conductor of

the impulse, whilst the heart and large vessels are formed of elastic tissues, which have little conducting power; moreover, the walls of such apparatus, when exposed to pressure, remain unchanged, whilst the walls of the heart shorten and contract."—I consider that the objections here stated by Kiwisch, have been already answered.

It sounds strange, to hear any one say that he rejects all explanation of the heart's impulse by physical laws. Is it possible to explain the impulse otherwise than by the laws of physics? or are there certain physical laws, to which the heart is not subjected? The only question for us to answer is this, whether the pressure is really powerful enough to produce a movement of the heart in a direction downwards and forwards. A gun does not recoil, if only a small quantity of powder be exploded in it; and Segner's wheel does not move, if the friction be great, and the column of water small. Now, observation shows us, that in many cases the heart, during its systole, evidently moves downwards with great rapidity, and is forced against the walls of the thorax.* In my opinion, this fact is only capable of explanation by the above-mentioned physical law.

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* On the 8th of March, 1846, I examined a child, a few days old, in whom the sternum was wanting, and whose thorax, in consequence, presented in front a cleft, narrow above and broad below, and closed in merely by the integuments. At each inspiration, the skin was forced backwards towards the vertebræ, and the anterior ends of the ribs thereby bent somewhat inwards; during expiration, the skin was pressed outwards, in the form of a bladder. It was readily ascertained by

In every case in which blood is forced out of the ventricles, it must be considered as a cause cooperating in the production of impulse.

I have never asserted that this was the only cause

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palpation, that the heart lay in a vertical direction, that during each systole it moved downwards and forwards, and at each diastole upwards and backwards. At each systole, in fact, the impulse of the heart could be felt immediately above the insertion of the diaphragm, and, at each diastole, on a level with the second rib, provided the finger was pressed deeply enough towards the vertebræ at that point. The impulse of the diastole was as strong as that of the systole. When two fingers were so placed, that the lower one could feel the systolic, and the upper one the diastolic impulse, it appeared as though the heart glided about an inch downwards at each systole. When the skin was moderately stretched between the fingers, the outline of the heart was seen as well marked during the systole, as during the diastole; from which it may be concluded, that the impulse felt at the points indicated, is not caused by any enlargement or lengthening of the heart, but by a sliding movement. When the integuments were not touched, the outline of the heart, during its systole, could be seen through the puffed-out integuments to form, at each expiration, an elevation passing from above downwards; whilst, during the diastole, a depression of the inflated skin was observed, passing from below upwards.

When the integuments were pressed backwards towards the vertebræ during inspiration, the contour of the heart became as distinct during the systole as during the diastole. When the child was placed upon its back, the movement of the heart downwards was in a direction closely corresponding to the centre of the cleft; when laid upon its side, the movement was somewhat diverted towards that side on which it lay. I do not think it is necessary that I should enter into any explanation of this case, for I should only have to repeat what has been already said.

of the heart's impulse. The double and triple impulse, associated with a single beat of the pulse, and with simultaneous contraction of both ventricles; weakness of the pulse, conjoined with strong impulse of the heart, when the condition of all its valves is normal; the slow heaving of the thoracic walls, unattended by concussion, and other extensive disturbances of the heart, occurring during its systole—are not explicable by this law.

Bouillaud relates cases, in which a double and even a triple beat of the heart was observed with each arterial pulse. He ascribed the second and third beat, not to a repetition of the systolic impulse, but to a diastolic impulse. It sometimes happens, when the heart is forced over to the right side by extensive effusion in the left pleura, that, at each systole, an impulse is felt at that part of the thorax which corresponds to the situation of the heart's apex; and that a second impulse, or rather a heaving of the thoracic walls, is also observed during the diastole, about an inch and a half above that part. The same phenomenon is also occasionally observed in cases of great hypertrophy, with dilatation of both ventricles: at each systole, the part of the thorax which corresponds to the heart's apex is raised, and the part over the middle of the heart falls in somewhat, whilst, during the diastole, the reverse of these movements is observed.

When the heart occupies a low position, in consequence of the ascending aorta being lengthened, the elevation of the thoracic walls, during the diastole, becomes very marked. The opinion of Gendrin, that

an impulse—particularly when of a heaving character—felt beneath the third rib during the ventricular diastole, is a sign of the heart being placed low, is quite correct. In many cases of hypertrophy with dilatation of both ventricles, the systole causes an impulse, but the diastole produces no heaving of the walls; a concussion, however, is felt, not excited by the impulse of the heart against the walls of the thorax, apparently synchronous with the retreat of the heart towards the vertebræ. This phenomenon is evidently the same as that described by Laennec, under the name of impulse of the auricles.

I have never observed a distinct impulse, *i.e.* such an one as takes place during the ventricular systole, associated with the heart's diastole. In the cases which I have seen, where there was a double or triple impulse of the heart accompanying each pulse, the impulse was always produced during the ventricular systole.

The double or triple impulse was caused in one of the following ways: either through a double or triple contraction of the right ventricle, united with a single contraction of the left; or by the two ventricles contracting alternately; or, in consequence of blood not being thrown into the aorta at each systole, its supply from the lungs failing, or the bicuspid valves being defective. Deficiency of the bicuspid valves, associated with single contractions of the heart, may be recognized by systolic murmurs heard in the left ventricle, and a faulty pulse; when the tricuspid valves are defective, a strong pulsation is observable in the veins of the neck, and a weakness of the sounds in the pulmonary artery.

I have never observed an impulse caused by contraction of the auricle. Bouillaud relates a case, where the left auricle was supposed to have produced an impulse; but it is not at all clear that the impulse was so produced. His grounds for the opinion were, that he could not explain the phenomenon observed in any other way. (*Traité des Malad. du Cœur*, Paris, 1835, t. i, p. 149.)

There are other causes, besides the above-mentioned, which assist in the production of the heart's impulse.

The lengthening of the arterial columns of blood, which occurs at each systole, must be particularly noticed. The blood contained in the arteries is not instantaneously driven onwards at each ventricular systole, so as to afford sufficient room for the additional quantity entering into them; but the arteries themselves enlarge in length and breadth, or, in other words, the arterial columns, at each systole, become thicker and longer: the lateral enlargement of the arteries is small, but their elongation considerable.

The aorta and pulmonary artery, being free and unattached to some distance from their origin in the heart, allow of a lengthening of the blood column downwards; and the heart will consequently be forced in that direction.

Dr. Messerschmid undoubtedly takes this view of the matter, but he gives no other explanation of it than this, that the resistance of the blood in the arteries is communicated backwards to the heart, through the blood contained in the heart, and thus

forces it downwards. The same explanation is also given by Gendrin, *Leçons sur les Maladies du Cœur*, p. 37.

We occasionally observe cases, where the heart's movement is slow, and where it descends as much as one or two inches lower during the systole, than during the diastole. Such a movement is inexplicable, except upon the supposition of the aorta being lengthened downwards; according to my own experience, indeed, such extensive movements of the heart only occur when the ascending aorta is longer than natural, but not widened.

A third cause of the heart's impulse, which I referred to, in the first edition of this work, is the change of the heart's form caused by its contraction. When the heart is much enlarged, the thoracic walls are sometimes extensively raised during its systole, and sink suddenly back during its diastole; and if the movements of the heart are slow, the heaving up of the thoracic walls may take place without producing any concussion. When the heart is enormously enlarged, I believe that it may raise the walls of the thorax, in consequence of its antero-posterior diameter becoming greater during the systole, than the space between the fore part of the thorax and the vertebral column; from which also it must be admitted, that the heaving of the thoracic walls, when no sign is present to demonstrate a simultaneous and equal pressure on the vertebral column, is only explicable by Dr. Gutbrod's theory.

Lastly, it must not be forgotten, that the heart, by becoming rigid from contraction during its systole,

takes a different form and direction from those it held in its relaxed state; and that such change of form and position may aid in producing its impulse, even though the idea of a lever-like elevation of its apex against the thoracic walls, be hardly explicable by the arrangement of its muscular fibres.

Dr. Kürschner—*Arch. für Anatomie*, etc., Von Johann Müller, 1841, vol. i, p. 103—offers the following explanation of the heart's impulse, as deduced from vivisections and post-mortem researches:—"The apex of the heart, during its diastole, is forced downwards by the flow of the venous blood, and the aorta and pulmonary artery consequently put upon the stretch. During the systole, the ventricles are freed from the pressure of the venous blood by the closure of the auriculo-ventricular valves, the extended arteries are shortened, and the apex of the heart raised. The reason of this raising of the apex being so forcible as to produce a sensible blow on the walls of the thorax, is, that the blood is driven in that direction by the powerful contraction of the muscular fibres, the heart itself acquiring great firmness and consistency."

I have not repeated Dr. Kürschner's experiments, and therefore am not able to say anything about them. This much, however, is very clear, that Dr. Kürschner, in his explanation, has not troubled himself much about the laws of mechanics.

If it be correct, that the stream of venous blood presses the apex of the heart towards the vertebral column during the diastole, the pressure must continue during the heart's systole, notwithstanding the

closure of the valves, for the pressure could not cease, unless the valves were of themselves sufficient to resist the influx of the venous blood from the auricles into the ventricles: but they possess no such power of resistance, and consequently the pressure of the venous blood on the ventricles, both during the diastole and the systole, will be alike in force and in direction.

According to Dr. Heine, the heart is thrown forwards by the contraction of the papillary muscles. The motion of the heart forwards and to the left is thus accounted for: the broad extremities of the mitral and tricuspid valves are fixed to segments of the fibro-cartilaginous aortic ring, in an oblique, not a perpendicular direction, and when suddenly stretched, throw the heart forwards; whilst the arteries, being attached laterally and superiorly, do not yield to the movement, but bend forward towards the base of the heart.

But, according to Dr. Heine's own showing, such an effect could not be produced, except upon the hypothesis that the contraction of the heart commences at the points of insertion of the papillary muscles, so as to afford these a firm point of support for their action; such contraction of the heart being followed by contraction of the papillary muscles, by which the heart, still undiminished in size, is thrown forward; then comes the heart's systole, and the expulsion of the blood.

I must say, that this explanation of the forward movement of the heart by the mode of insertion of the papillary muscles, and their contractions, is not

very intelligible to me; if such an explanation is admitted, the heart's impulse can no longer be looked upon as a concomitant circumstance, but must be regarded as the very object of its action—an idea to which I must refuse to give my assent. The object of the contraction of the heart is to drive the blood onwards; and, in my opinion, we have good right to take this fact as a postulate. In explaining the cause of the heart's impulse, we must proceed by the process of demonstration; we have no *à priori* facts to aid us.

I might add, that, according to experience, the force of the heart's impulse bears no relation in degree to the development of the papillary muscles. Dr. Heine has not taken into consideration the slowly-developed impulse of the heart. The explanation of the movement of the heart downwards, as a consequence of the contraction of the papillary muscles, can scarcely be entertained as serious.

Professor von Kiwisch gives the following explanation of the cause of the impulse: "The heart cannot recede from the diaphragm and the thoracic walls, unless some body—either gaseous or liquid—or a portion of lung, intervene between the two. Now it is evident, that at those parts where the pericardium is attached to the thoracic walls and to the diaphragm, no lung can intervene; so long therefore as the pericardium contains neither liquid nor gaseous bodies, the thoracic walls and the diaphragm form the fixed points of the heart; the other parts around the heart are yielding, and follow its movements; the heart, consequently, during its diastole,

will be lengthened, and take a direction upwards and backwards. The ribs alone form the rigid portion of the thoracic walls, the intercostal spaces being more or less yielding; when therefore a contraction of the heart as it lies in contact with the walls takes place, the ribs, being the most unyielding parts, form the fixed points against which the organ is firmly pressed, and from which it cannot recede.

"In this fixed position, the heart, during each systole, swells out, becomes firmer, and takes a more globular form; its movement forward, thence resulting, is restrained by the ribs, but manifests itself through the yielding intercostal spaces; and thus it is, and by no other means, that the phenomenon in question of the heart's impulse is produced. Accordingly, when we place a finger in the proper intercostal space, we do not feel what is wrongly called the beat (*Anprallen*) of the heart's apex against the thoracic walls, but we experience a sensation as though the heart's walls were hardening and swelling, being themselves fixed and at rest; much the same sensation, in fact, as is experienced when the hand is passed through the abdomen and placed upon the contracting ventricles of the heart, the diaphragm intervening, or when the thorax is opened and the hand laid immediately upon the heart."

The following remarks may perhaps serve to explain this theory of Kiwisch.

The heart cannot recede from the walls of the thorax and the diaphragm unless some foreign body intervene, because the formation of a vacuum is prevented by the pressure of the atmosphere; this pres-

sure acts upon the position of the heart only through the medium of the lungs; and the heart cannot recede from the thoracic walls and the diaphragm, unless the lungs are distended with air. The lungs possess elasticity and organic contractility, by which a continual opposition is offered to their distension by the air; their contractile power is the measure of the force, by which the soft parts of the thorax are drawn inwards. When the abdomen is opened, the diaphragm is found to be arched upwards, and the intercostal spaces—provided the individual examined be not unusually fat—are seen to be furrowed externally, and elevated within the thorax. This drawing-in of the intercostal spaces is not confined to those parts of the thoracic walls beneath which the lungs lie; the pressure of necessity acts upon every part of the thoracic walls, and equally well, therefore, whether through the heart, through pleuritic effusions, or through an infiltrated portion of lung. The pressure is constant, but it is increased by inspiration. Since the heart is retained in contact with the thoracic walls and the diaphragm, solely by the distended lungs, and since the contractility of the lungs produces a constant drawing-in of the soft parts of the thoracic walls, it follows, that whatever form the heart may take, it can never cause a projection of the intercostal spaces, or of the diaphragm, in a direction outwards or downwards; if no other influence affected the heart's position, a slight drawing-in of the intercostal spaces, and the diaphragm, would be more likely to happen at each systole.

The theory of Professor von Kiwisch pre-supposes

that the lung both contracts inwardly, and presses outwardly. If the explanation above given does not make the matter clear to any one, let him recollect, that at times the impulse of the heart not only forces forward the intercostal spaces, but also raises the sternum and the ribs; a fact which has been quite overlooked by Kiwisch.

The force and extent of the heart's impulse.

A consideration of the causes which produce the heart's impulse enables us, in some cases, to premise certain facts respecting its force and extent.

The different causes referred to in the preceding account are not always found associated together in the production of the heart's impulse; one only may be in operation, or there may be several; the force of the impulse is necessarily connected with the rapidity and completeness of the contractions, and with the size of the organ itself.

If, for the moment, we admit that the pressure of the blood on the walls of the ventricles, and the lengthening of the arterial columns, are the only causes of the heart's impulse, it follows that this impulse will be stronger, in proportion to the quantity of blood and the velocity with which it is forced from the heart into the arteries. An hypertrophied and dilated ventricle is therefore peculiarly well fitted for the production of a strong impulse. In simple hypertrophy, without dilatation, as well as in dilatation of the ventricles, without the thinning of their walls, the impulse is weaker than in hypertrophy with dilatation, but stronger than in the normal con-

dition of the heart; on the other hand, when its cavities are dilated, and its walls thin, it is weaker than in the last case, because a simply dilated ventricle cannot completely expel the blood within it. An hypertrophied and contracted ventricle produces a diminished impulse. The more contracted the ventricle, the weaker is the impulse.

But we must not conclude, that in hypertrophy with dilatation the impulse is always stronger than in hypertrophy without dilatation; for rapidity and completeness in the heart's contractions are indispensably required for the production of a strong impulse, and these, as experience teaches us, depend upon other causes, than merely the thickness of the heart's walls.

An hypertrophied heart, when excited, may produce strong concussion of the thorax; and yet, at another time, beat so calmly that its impulse is almost imperceptible.

If the hypertrophy and dilatation is confined to the left ventricle, and the right ventricle is either of normal size, or smaller than natural, the left ventricle will be unable to throw its full quantity of blood into the aorta, on account of its receiving a deficient supply from the right ventricle, which does not contract more frequently than the left. Hence, no distinct and constant increase of the heart's impulse will attend hypertrophy and dilatation confined to the left ventricle, provided the aortic valves are not defective; for when they are, a portion of the blood which is driven into the aorta during the systole, regurgitates into the ventricle during the diastole;

from time to time, however, a beat more violent than ordinary will be observed. The same is true of hypertrophy and dilatation of the right ventricle, accompanied by normal or diminished size, with thinning of the walls of the left ventricle, provided there be no defect of the tricuspid valves; the greater the disproportion in size between the two cavities, the less will be the force of the impulse.

The force of the impulse is also affected by the size of the arterial opening, and the quantity of blood contained in the ventricle. If the arterial opening be narrow, and the ventricle large, the impulse of the heart is less than it would be if the ventricle and arterial opening were both enlarged: when the arterial opening is narrow, the impulse is prolonged, if the contraction of the ventricle be complete. If the disproportion between the size of the arterial opening and the ventricle be considerable, the heart cannot contract thoroughly, its impulse is shortened, and may be scarcely perceptible, although the organ be hypertrophied and dilated.

But it has been shewn that the impulse of the heart may be caused by mere change of its form, independent of any expulsion of blood from its ventricles; consequently, what has been here said respecting the effect which the condition of the arterial openings and the ventricles has upon the impulse, must be taken in a modified sense. Change of form does not produce any increased impulse, unless the heart be much enlarged and its action violent.

The heart's movement may be considerable, and yet produce no concussion in the walls of the thorax;

and the more distensible the aorta and pulmonary artery are, the greater is the movement produced by the ventricular systole. The greatest displacement of the heart occurs (as we have already said) when the ascending aorta is lengthened without being widened. As a general rule, lengthening of the aorta is only observed in old persons. Little or no lengthening of the aorta takes place, when blood is forced into it during the ventricular systole, provided it be not widened. In such case, there is either very slight, or no downward movement of the heart at all. A rapid contraction of the organ is not absolutely indispensable to the lengthening of the aorta; but the lengthening is always greater, the greater the quantity of blood driven out of the ventricle.

The Direction in which the Heart moves during its Systole, and the Part where the Impulse is felt.

Both the ventricles must contract synchronously, for we find that, with very rare exceptions, one beat of the pulse takes place at every impulse of the heart; if this were not the case, a double impulse of the heart would accompany a single pulse. It is not a very easy matter to determine the direction which the heart takes during the contraction of its ventricles; the contraction of the right ventricle forces it in a direction different to that which the left ventricle would give it; their simultaneous contraction consequently produces a movement in the diagonal of the forces. When the heart is in its normal position, its impulse may be felt between the cartilages

of the fifth and sixth ribs of the left side; if it lie in a vertical position behind the sternum, it is forced downwards at each systole, and consequently strikes against the lower part of the sternum, or at the scrobiculus cordis; in the latter case, the parts are seen to rise during the systole, and to return to their former state during the diastole.

When the position of the heart is horizontal, from right to left, its impulse will be felt in the intercostal spaces of the lower true ribs on the left side.

Its vertical position is not observed, except in cases of extensive pleuritic effusions, or in pneumothorax of the left side, or of vesicular emphysema affecting the lower part of the left lung, or the whole of the left lung, or both the right and left lungs together.

The horizontal position is given to the heart, either from the diaphragm being forced upwards on the left side by abdominal effusions, or by gaseous distension of the bowels; or through enlargement of the left lobe of the liver; or through extensive pleuritic effusions, or pneumothorax of the right side, by which the right lobe of the liver is depressed and forced towards the left, the left lobe being thereby driven upwards. This position of the heart may be also produced by its own enlargement (the diaphragm retaining its normal condition), by lengthening of the ascending aorta, or by large aneurisms on the right side of the ascending aorta; the further the apex of the heart is pressed towards the left, the more extensive must the abnormal conditions be which cause the pressure.

Laennec, and other writers after him, tell us, that when the left ventricle is hypertrophied, the impulse of the heart is felt in the left side, but when the right ventricle is hypertrophied, behind the sternum. This statement is by no means correct. If the heart occupies the vertical position, its impulse is felt behind the sternum, whether its right or its left ventricle be hypertrophied; but if it be placed horizontally, the impulse is felt in the left side, even though the right ventricle be hypertrophied. When the heart is considerably enlarged, its impulse may be felt behind the sternum, at the pit of the stomach, and in the left side.

Different Degrees of Force in the Heart's Impulse.

Three several degrees may be noted in the force of the heart's impulse, each, however, passing gradually into the other,

1. An impulse which does not raise the thoracic walls, nor shake the head of the auscultator; or which is imperceptible. The heart producing such an impulse may be perfectly normal, or it may be more or less hypertrophied and dilated, or dilated only; the hypertrophy and dilatation may be confined to one ventricle, the opposite state of things obtaining in the other; the impulse, lastly, is affected by the various degrees of pericardial effusions. From all which it results, that a weak or imperceptible impulse is in itself a very indefinite sign.

2. An impulse which does not raise up the thoracic walls, but imparts a strong concussion to the head of the auscultator. Such an impulse indicates hyper-

trophy of one or both sides of the heart, its cavities either remaining of normal capacity, or being slightly dilated; or it may be the result of increased action in a healthy heart. We can only determine upon which of these causes it depends, by ascertaining the size of the heart. If the heart be of normal size, its walls are not hypertrophied, and the strong impulse is the result of increased action; but if it be enlarged, and communicate a concussion to the head of the observer, there can be no doubt that its walls are hypertrophied.

3. An impulse which raises the thoracic walls during the heart's systole, the walls sinking again during its diastole. The head of the auscultator necessarily follows these movements. The heaving of the thoracic walls takes place either suddenly, producing a concussion, or the movement is gradual, and unaccompanied by concussion; in the latter case, the heaving may be scarcely remarked by the auscultator, but the collapse, from its suddenness, becomes the more striking. In consequence of a concussion being communicated to the head when the heart sinks back, it is not impossible that a person little versed in auscultation might confound the sinking back with the impulse, and thus mistake the diastole for the systole.

An impulse strong enough to raise the thoracic walls and the head of the auscultator, requires for its production hypertrophy and dilatation of both ventricles. Hypertrophy and dilatation of the left ventricle only, will not produce such an impulse, unless there be considerable defect of the aortic

valves co-existing; the same remark will apply to the impulse which raises the thoracic walls without imparting concussion to the head of the observer. A slow contraction of the heart is the consequence, either of a narrowing of its orifices, or of the area of its cavities preponderating over the thickness of their walls, or of a general deficiency of the blood, etc.

The second, as well as the third degree of impulse, does not preclude the idea of pericardial effusion, or of union between the free and attached pericardial surfaces. It is only when the effusion is considerable in relation to the hypertrophy of the heart, that the increased impulse is lost; when the heart is attached to the free pericardium, and the layers of matters forming the union are thick, the thoracic walls may be raised by an impulse similar to that produced by an hypertrophied and dilated heart: the simply hypertrophied heart never produces a concussion.

The impulse moreover varies, both as to the extent of surface and the parts over which it is felt. These points can be much more readily determined by palpation, than by auscultation. The impulse of a normal heart cannot be felt in more than one, or at most in two, of the intercostal spaces; if it be perceptible in several, or through more than an inch and a half of one, at the same moment, we may conclude that the heart is enlarged. The cause and the signification of the heart's impulse, as felt at different parts of the thorax, and at the pit of the stomach, have been already pointed out.

II. THE PULSATION OF THE ARTERIES.

We shall only speak here of the pulsation of the aorta and pulmonary artery. If the ascending aorta, or its arch, be so enlarged, as to come in contact with the thoracic walls, or if any tumour be present in the anterior mediastinum, an impulse will be observed, at each systole of the heart, over those parts of the thorax which correspond to the course of the aorta—an impulse as strong, or even stronger, than that of the heart itself.

But we cannot be certain that the impulse is produced by pulsation of the aorta, unless it is accompanied by a perceptible heaving of the thoracic walls; for the concussion produced by the impulse of the heart is often distributed over a considerable extent of surface, particularly over the sternum. When we are thus in doubt as to the cause of the beat felt at the upper part of the sternum,—whether it be produced by the pulsation of the aorta, or by the heart's impulse,—we must carefully examine the parts, proceeding from above downwards to the heart's apex, and compare the force of the concussion as felt at different points; if it be stronger towards the upper part of the sternum, than at any point nearer the heart's apex, then it is evident that the beat is caused by pulsation of the aorta. It is however advisable in all cases to aid the diagnosis by percussion.

The pulsation of the pulmonary artery may be observed over the thorax, if a consolidated portion of lung, or any solid body, intervene between its root,

or one of its large branches, and the thoracic walls. Hepatization, or tubercular infiltration of the upper lobe of the lung, are the most frequent causes of this phenomenon, which Laennec attributed to transmission of the heart's impulse.

I have never felt the pulsations of the descending thoracic aorta. The pulsations of the abdominal aorta are readily felt in thin persons, when the abdomen is retracted; in such cases there is no difficulty in discovering any existing enlargement of this vessel.

III. THE SOUNDS AND MURMURS HEARD IN THE REGION OF THE HEART, AND OVER DIFFERENT ARTERIES, CONSEQUENT UPON THE HEART'S MOVEMENTS.

The normal sounds of the heart are generally indicated by the expression "tic-tac"; its abnormal sounds being comprised under the terms of bellows, sawing, rasping, filing murmurs, etc. The tic-tac may be stronger or weaker than natural, or altered in its timbre; we are therefore obliged to speak of over-strong, or over-weak, or too ringing—and consequently abnormal—normal heart sounds. This tic-tac I call the sounds (*Töne*) of the heart, and speak of normal and abnormal sounds. By murmurs (*Geräusche*) I understand the abnormal sounds of the heart indicated above, blowing, sawing, rasping, etc.

Gendrin gave the name of *bruit de choc*, and *bruit de percussion* to the tic-tac, to distinguish it from the blowing, sawing, etc., murmurs. I believe that the term chosen by myself is preferable.

THE SOUNDS OF THE HEART.

Cause of the Sounds.

Observers are by no means agreed as to the interpretation of the origin of the two sounds which are heard over the region of the heart, and are synchronous with its systole and diastole. Laennec attributed the first prolonged sound to contraction of the ventricles, and the second to contraction of the auricles, but did not enter into any particular explanation of their mode of origin; doubts were afterwards thrown upon this opinion, through the researches of Haller, who showed that a short and rapid contraction of the auricles preceded—*wie ein Vorshlag*—the systole of the ventricles.

Majendie attributed the first sound to the impulse of the heart's apex, and the second to the impulse of the anterior surface of the right ventricle, against the walls of the thorax.

At each systole, some part of the heart, if not in every case its apex, beats against the thoracic walls, the impulse frequently giving rise to a ringing metallic sound, which may be imitated by tapping upon the back of the hand laid flat over the ear. But the beat of the heart against the thoracic walls is not the only cause of the first sound, for the sound may be quite distinct when the impulse is scarcely perceptible, and on the other hand it may be barely audible when the impulse is violent. Moreover, it is shown by vivisections, that the sounds of the heart continue, although the organ be prevented from striking against the sternum, or any other body.

The right ventricle again does not strike against the thorax during the diastole, at least in the normal state of things.

Rouanet attributed the first sound to a stretching of the auriculo-ventricular valves, during the ventricular systole; and the second to the distension of the semilunar valves, produced by the backward pressure of the blood in the arteries during the heart's diastole.

Rouanet considered his views supported by the fact, that membranes and chords produce a sound when suddenly rendered tense; this fact he applied to the valves of the heart, which are rapidly brought into a state of tension during the alternate systole and diastole of that organ; and he endeavoured experimentally to demonstrate the correctness of his theory. For this purpose he tied a glass tube four feet long into the aorta above the semilunar valves, and beneath the valves he fixed a short tube, having a bladder filled with water attached to it; he then compressed the bladder, so as to force the water into the tube fixed above the valves, and suddenly relaxed the pressure; at each descent of the fluid, he noticed a stroke or sound which bore a certain degree of resemblance to the second sound of the heart.

Professor Bouillaud concurred in Rouanet's theory, giving the name of valvular sounds to the ordinary heart sounds. In addition to the arguments used by Rouanet in support of his views, Bouillaud brought forward this important one, viz.: that the sounds of the heart are but very little affected by change in its structure, so long as the valves duly

perform their functions; but that they undergo constant and remarkable changes, and are converted into sounds of a totally different character, whenever the valves are altered in structure. He was not, however, of opinion, that the first sound depended entirely upon the distension of the auricular valves during the systole, but attributed it in part to the sudden flapping back of the semilunar valves against the walls of the arteries; nor did he consider that the second sound was produced solely by distension of the semilunar valves, but also in part by the simultaneous stroke of the auricular valves against the walls of the heart, at the time the blood rushes from the auricles into the ventricles.

Dr. C. J. B. Williams at first concluded, from the results afforded by vivisections, that the first sound was a muscular sound; but he subsequently attributed it to the vibrations excited in the ventricular walls and the auricular valves during the heart's contractions; the second sound he explained by the impulse of the blood against the semilunar valves.

The following are the conclusions drawn by the Dublin Committee from their vivisections:—

1. The sounds are not caused by contact between the ventricles and the sternum, but by movements in the heart and its vessels.

2. The distinctness of the sounds is increased by contact of the ventricles with the sternum and anterior part of the thoracic walls.

3. The first sound is connected with the systole of the ventricles, and is of equal duration with it.

4. The cause of the first sound comes into action

and ceases with the systole of the ventricles, and is constantly in action during the continuance of the systole.

5. The first sound does not depend upon the closing of the mitral and tricuspid valves, their act of closure being effected at the commencement of the systole, and being of much shorter duration than it.

6. The first sound is not produced by friction between the internal surfaces of the ventricles, for friction can only take place when the ventricles are empty, whereas the first sound commences with the systole.

7. The first sound is caused either by the sudden rush of blood over the irregular internal surfaces of the ventricles during its passage towards the arterial openings, or by the muscular sound of the ventricles, or probably it is the result of a combination of these two causes.

8. The second sound commences immediately on cessation of the systole, and the integrity of the semilunar valves is necessary for its continuance; it seems to be caused by the sudden stoppage, through the action of these valves, of those movements of the blood which are occasioned by the elasticity of the arterial trunks, and occur after each contraction of the ventricles.

The Committee close their report by observing, that, notwithstanding the numerous researches which had been made, the subject was not yet exhausted, and that many doubtful points still required explanation.

According to Gendrin, the contraction of the ven-

tricles produces undulations in the blood within them, which undulations converge towards the heart's apex, and are communicated to its walls; and hence the origin of the first sound. It is loudest at the spot where the apex strikes the thoracic walls, partly because the undulatory vibrations converge towards this point, and partly because the sound passes most readily from the apex to the walls of the thorax, in consequence of the apex coming in contact with the walls.

At each diastole the blood rushes into the ventricles, first flowing down towards the heart's apex, then upwards, striking at last against the walls of the heart, about its base. This impulse gives rise to the second sound, which consequently is heard loudest at the heart's base; the semilunar valves have no part in its production, being already closed before the ventricle is filled, and thus before the second sound commences. Neither do the auricular valves assist in the formation of the first sound, for their vibrations must be mixed up with the vibrations of the blood: the first sound, moreover, is heard loudest towards the heart's apex, and not in the neighbourhood of the auricular valves; were it dependent upon the tension of these valves, it ought to be absent in cases where they are thickened or partially destroyed, but it is often heard loudest in such cases. And so again, destruction of the semilunar valves should put an end to the second sound, if the sound depended upon them; but the sound is always present, though masked by abnormal murmurs, which prevent its being recognized by the auscultator. Any one may

convince himself of the presence of the sounds, by listening for them, when the ear is removed to a little distance from the thorax.

Cruveilhier considers that both sounds originate about the roots of the aorta and pulmonary artery; the first being caused by the flapping back of the semilunar valves, the second by their closure.

His views were formed from observations made on an imperfectly formed infant. The heart of the child (which was in other respects well developed and full of life) lay outside the thorax, passing through a round opening in the upper part of the sternum. It was uncovered by pericardium, and completely bare, its colour pale, and surface dry. Its position changed when the child's posture was altered; when the child was placed vertically the heart sunk considerably, and its great vessels became visible. The axis of the heart was vertical. Its action was not disturbed either by touching or by gentle pressure, nor did the manipulation give rise to pain. Both its sounds were heard when the ear was laid immediately on its surface, the first sound much weaker than as ordinarily heard through the thoracic walls. Both sounds were loudest at the base of the heart, and weakest at its apex. To ascertain the cause of the first sound, Cruveilhier examined every part of the surface of the ventricles, but could discover there neither vibration nor sound, which could be looked upon as other than the consequence of conduction. From this he concluded that the auricular valves yield no sound, and that the first sound is caused by the flapping back of the semi-

lunar valves, being heard loudest at the same spot as the second sound. He considered his views supported by clinical observations; the sounds of the heart, according to him, being altered under every diseased condition of the semilunar valves, when the auricular valves are healthy. The impulse of the heart against the thoracic walls assists in the production of the first sound, and this is the reason why the first sound is loudest at the heart's apex.*

The Author's views respecting the Cause of the Sounds of the Heart.

The ventricles, the aorta, and pulmonary artery, severally contribute in the production both of the first and of the second sound of the heart.

The question, as to the origin of the heart's sounds, cannot, in my opinion, be solved by vivisections alone; observations must also be made upon individuals both in health and in disease, and a careful comparison be instituted of facts observed during life, with the results found after death.

A person practised in auscultation, with sufficient opportunities for investigation at his command, will find the following statements correct: the sounds which depend upon the movements of the heart differ in healthy individuals in their degree both of strength and of clearness; in one person they are indistinct, or barely perceptible, in another remarkably clear, having even a ringing character; at one time the sounds are scarcely audible in the region of

* *Gazette Médicale de Paris*, 1841, No. xxxii.

the heart, at another distinctly so over the whole anterior surface of the thorax, and even reach to the back; again, in some persons they are particularly clear at those parts of the thoracic walls against which the heart beats, whilst in others they are indistinctly heard there, but are very clear over the pulmonary artery and aorta.

By comparing the sounds heard over those parts of the thorax against which the heart beats, with the sounds heard above the base of the heart, where the pulmonary artery and aorta lie, we frequently observe that the first sound—the sound synchronous with the beat of the heart—is more prolonged over the heart than the second sound, but that above the base of the heart the accent falls on the second sound.

The sounds heard over the apex of the heart—and consequently over the left ventricle—also frequently differ, in strength and clearness, from the sounds heard at the same level, to the right and over the sternum—that is, over the right ventricle; I have also occasionally observed a difference in their pitch.

The sounds heard above the base of the heart—about the middle of the sternum, and towards its right border, beneath which parts the aorta passes—also occasionally differ in strength and clearness, and in some particular cases in pitch also, from the sounds heard at the same level, but about an inch to the left of the sternum.

These differences of sound are much more distinctly marked in individuals suffering from diseases

of the heart than in the healthy; it will be well, therefore, for observers to commence their investigation of them in persons suffering from disease, and afterwards study them in healthy subjects.

Whoever has extensive opportunities for observation of diseases of the heart, will meet with cases where neither the first nor the second sound can be heard at that part of the thoracic walls against which the apex of the heart beats—that is, over the left ventricle—but in their place he will find a single or a double murmur, blowing, sawing, rasping, etc.; while, at the same time, to the right of this,—over the right ventricle, and above the base of the heart, *i.e.* over the pulmonary artery and aorta—both sounds are distinctly audible; the sounds, moreover, heard at the three points indicated, generally differ from each other in strength, clearness, etc. In other cases again, both sounds, which in most cases differ from each other, will be heard over the left ventricle, the aorta, and the pulmonary artery, a murmur, synchronous with the ventricular systole, being audible over the right ventricle, but no sound.

Still more frequent are the cases in which a single or double murmur, but no sound, is perceptible along the course of the aorta, whilst over the right and left ventricles, and over the pulmonary artery, both sounds are distinctly audible. A single or a double murmur may also be heard over the left ventricle and the aorta, the normal sounds still existing over the right ventricle and pulmonary artery; or murmurs may be heard over the left and right ventricles, or over the right ventricle and the aorta, or over the

left and right ventricles and the aorta—whilst at those parts where no murmur is heard, the sounds are either distinct, or indistinct, or altogether inaudible.

If these remarks are correct, and I believe them to be so, inasmuch as they are the results of a vast number of observations made by myself and others, it appears to me tolerably clear that the ventricles, the pulmonary artery, and the aorta, severally assist in producing the sounds of the heart.

Variations in the sounds are generally associated with changes in the condition of the valves of the heart; we must, therefore, in our interpretation of these sounds, take into consideration the state of the valves during the heart's movements. From observations made on the living, and compared with post-mortem appearances, we are forced to the conclusion that variations in the sounds and murmurs of the heart are generally associated with changes in the condition of its valves; for when we observe during life that the sounds are replaced by murmurs, as a rule, we find an abnormal state of the valves after death; they are thickened, or contracted, or covered by excrescences, or the opening they enclose is constricted, etc. It must, nevertheless, be admitted that we occasionally remark during life no change of sound, or only such as is consistent with the healthy condition of the valves, in cases where an abnormal state of the valves is found after death. From which it follows, that not every abnormal condition of the valves gives rise to well marked variations in the sounds, but that certain particular abnormal states

are necessary for their production; or else, that other circumstances, besides these abnormal states of the valves, assist in producing them.

By endeavouring to obtain a clear idea of the manner in which the valves conduct themselves during the movements of the heart, both in their normal and abnormal states, we may perhaps obtain an insight into the conditions under which the valvular sounds, and their changes, and their conversion into murmurs, are produced. Such a survey of the possible conditions under which these changes may be brought about, would afford us guidance in our inquiries, and thus, by aid of observations and direct experiments, we may be able to separate facts from mere conjectures.

State of the Mitral and the Tricuspid Valves during the movements of the Heart.

Laennec asserted that the papillary muscles were so connected with the valves that their contraction necessarily caused the valves to open; he must, therefore have supposed that the papillary muscles do not contract synchronously with the substance of the heart, but during its diastole, *i. e.* when the blood enters into the ventricles. Bouillaud, on the contrary, maintains that the valves are closed by the contraction of the papillary muscles.

Now it will be found that the valves do not close, however forcibly the papillary muscles and the tendons attached to them are stretched in the direction which they hold in the heart, and that whether the force used be great or small, the valvular opening is

not diminished. The closure of the valves is therefore not produced by shortening of the papillary muscles during their contraction: neither do we observe that the blood meets with any obstruction in flowing from the auricles into the ventricles when these muscles are relaxed; hence their functions do not seem to be such as either Laennec or Bouillaud attributed to them. But since the contraction of these muscles cannot close the valves, their closure must necessarily be effected by the pressure of the blood against them. The office of the tendons, which pass from the muscles into the valves, is evidently to prevent their eversion; for if the free borders of the mitral and tricuspid valves were not firmly held by the tendons which are inserted into them, these valves, during the ventricular systole, would be driven by the stream of blood partly into the auricles and partly against the arterial openings, and anything like closure of them would be quite impossible.

The tendons are affixed to the valves in a manner which is of the highest importance to the correct performance of their functions; without such adjustment, indeed, the mitral and tricuspid valves could not prevent the reflux of the blood from the ventricles into the auricles during the heart's systole.

Several strong tendinous cords pass from each papillary muscle, towards the middle of that surface of the valve which is turned towards the ventricle, and are there inserted; some of them run on towards the base of the valve, and are inserted near the point of union of the valve with the walls of the heart. Now from these strong tendinous cords, somewhere

about their middle, and in part also from the papillary muscles themselves, smaller cords arise, and are inserted somewhat nearer to the free border of the valve. Still finer cords again spring from these last, and are inserted yet nearer to the free border of the valve, and into the border itself. No tendinous cord is attached to that surface of the valve which is turned towards the auricle.

If the papillary muscles be stretched in the same direction as that which they take in the heart, it will be seen that merely the stronger tendinous cords,—those which spring from the muscles themselves,—are rendered tense; the finer, which take their origin from the stronger tendinous cords, and are inserted nearer to the free border, or into the border itself of the valve, remain lax, however strongly the muscles may contract. Consequently, the free border of the valve is never rendered tense, by contraction of the papillary muscles, but only that portion of the valve included between its fixed border and the part, where the tendinons cords springing from the papillary muscles are inserted; the remainder of the valve, the part included between its free border and its middle, remains relaxed.

When any point of this relaxed surface of the valve is pressed in the direction of the auricle, so that the tendinous cords inserted into it, are made tense, it will be seen that a number of pouches are thereby formed; and if the same experiment be tried over the whole of the valve, the ventricular surface will lose its smoothness, and be thrown into a series of folds, which commence at the free border

of the valve, and reach as far as, or even further than, the middle of the valve; these folds are evidently formed by the peculiar insertion of the tendinous cords. Again, by blowing against the relaxed portion of the valve, in a direction towards the auricle, the valve will be made to swell out like a sail, and to exhibit the folds around its free border. And the same thing happens, when water is poured against it.*

Now, since the blood during the ventricular systole, has a tendency to flow back towards the auricle, it must necessarily be caught in these pouches, or semilunar-like swellings of the mitral and tricuspid valves, and thus cause the lax portions of the valves —the portions unaffected by the contraction of the papillary muscles—to project towards the auricle, so far, at least, as the tendons inserted into them will permit. Through the formation of these projections in the valves, the blood itself becomes the cause of

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* Dr. Kürschner imagined that, in my experiments, the border of the valve had not been completely unfolded, and that false folds had thus been formed in it. In their normal state, the tendinous cords—and particularly those of the mitral valves —do not end as such, but spread out into a kind of membrane, and are inserted into the valves in such manner as to form crescentic membranous folds. Now, if blood or water be forced between the membranous folds formed by the insertion of the tendinous cords and the valve, a bladder-like projection will be observed on that surface of the valve which looks towards the auricle: it is to this projection that I apply the term pouch (*Tasche*). These pouches are smaller in the tricuspid, than in the mitral valves, but they are always present under normal circumstances.

the complete closure of the passage into the auricle, *i.e.*, when the valves are held in such a position, that no opening remains after these projections are formed. It is therefore evident, that the mode of attachment, and the length of the tendinous cords of the mitral and tricuspid valves, cannot be fortuitous.

The area of the cavity of the ventricle at the commencement, is different from its area at the end of the systole; and the points of origin of the papillary muscles approach continually nearer to the attached borders of the mitral and tricuspid valves, as the systole progresses. Now, the length of the tendinous cords always remaining the same, it becomes evident, that for the perfect closure of the valves, the tendinous cords which retain the valves in their proper position, must of necessity take their origin from the papillary muscles.

If, for example, they arose immediately from the walls of the heart, and were of due length for the performance of their functions at the commencement of the systole, they would necessarily be of too great a length during its progress; and on the other hand, would impede the diastole, if their length was sufficient merely to retain the valves in their proper position towards the end of the systole. But as the length of these cords is invariable, they must necessarily have some muscular attachment; and the object of the papillary muscles is evidently, by alternate extension and contraction, to retain the valves in their proper position. During the progress of the systole, these muscles contract, the distance between their points of origin and the attachment of the mitral and tri-

cuspid valves continually diminishing; in consequence of this contraction, the tendinous cords arising from the muscles retain the same degree of tension during the progress of systole, which they had at its commencement, and they also retain it during the diastole, the papillary muscles being lengthened when the heart expands.

The correctness of the views here offered of the function of the papillary muscles, seems to me confirmed by the fact, that that division of the tricuspid valves, which lies against the heart's septum, receives its tendinous cords either from very short papillary muscles, or immediately from the walls of the heart. The points of origin of these tendinous cords approach but little, or not at all, during the systole, to the points of attachment of the valve; and recede as little during the diastole. Now here, a simple tendinous cord suffices for the proper restraint of the valve, because change in the length of the cord is not required.*

From what has been said, it would appear, that the movements of the mitral and tricuspid valves are as follows. During the systole of the ventricles, the papillary muscles contract, and prevent the valves from being forced out of the ventricles, and also restrain their movements in the direction of the arterial openings. At the same time, the papillary muscles, and the tendinous cords arising from them, approach

* The use of the papillary muscles, as here given, has been already pointed out by Professor Weber, in his edition of Hildebrandt's *Anatomie.*

each other, and thus the surface of the valves, into which they are inserted, is thrown into folds, and the valvular opening diminished in size.

The remainder of the valvular opening is closed by that portion of the valve which is not rendered tense by the shortening of the papillary muscles. The pressure of the blood, in fact, upon this portion of the valve, bellies it out like a sail; and opposite points of the free borders of the valves, thus swollen, come into contact and materially support each other; partly in consequence of this support, but still more through the attachment of the tendinous cords, the eversion of the free borders of the valves is prevented. Since the finer cords, which run into the free borders of the valves, spring from the larger tendinous cords, which have their origin from the papillary muscles, the pressure of the blood against the distended portion of the valves causes all the tendinous cords, arising from the muscles, in consequence of the attachment of the finer cords to them, to approach each other, and thus to take a curved direction.

At each diastole, the papillary muscles become lengthened, and separate from each other, and the blood, flowing from the auricles, would naturally press the valves against the walls of the heart, and also against the arterial openings, if they were not retained in their proper position by the tendinous cords. These tendinous cords, arising from the papillary muscles, are not relaxed, even during the diastole; for if they were, the valves could not, at the commencement of the ventricular systole, be brought into the position requisite for instantaneous

closure; and a great part of the blood would consequently, at each contraction, flow back from the ventricle into the auricle; the valves must indeed be frequently drawn into their proper position, by the contraction of the papillary muscles, in opposition to the current of blood.*

For the perfect performance of their functions, the free borders of the mitral and tricuspid valves must form the pouches above described, and the tendinous cords and papillary muscles must be of a length proportionate to the size of the ventricles. If the valves do not possess their normal conformation, they either permit a reflux of blood from the ventricle into the auricle, during the systole—*i. e.*, the valves are defective, or they present some obstacle to the entrance of the blood into the ventricle during the diastole.

In the first case, the abnormal condition is caused by thickening or shortening of the free borders of the valves, or by the union of their free borders with the tendinous cords attached to the middle of the

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* Dr. Kürschner thinks that he has discovered certain muscular fibres, which pass from the auricle into the valves, and attach themselves, either immediately or by tendinous cords, to the points of insertion of the tendons of the papillary muscles; not, however, to the larger tendinous cords—the tendons of the first order—but to those of the second order, namely, such as are inserted towards the free border of the valves. The object of these muscles is, to increase the distance between the valvular flaps and the tendons of the first order, during the contraction of the auricle, and to bring them into such a relation to the margin of the auricle, as to cause them, when pushed forward and distended, to close the auricular opening. My own opinion is, that such a contraction of the valves would serve no purpose whatever.

valves, so as to prevent the formation of the pouches; or by shortening, lengthening, or rupture of the tendinous cords; or by excrescences, or deposition of blood-coagula, etc., upon the edges of the valves; or by union of the surfaces of the valves with the walls of the ventricle.

In the second case, the abnormal condition is produced by extensive deposition of foreign matters, blood-coagula, or chalky concretions, etc., upon those surfaces of the valves which are turned towards the auricle; or by union of the tendinous cords among themselves, or with the free border of the valves, by which their perfect contact is prevented.

Action of the Semilunar Valves.

The semilunar valves of the aorta and pulmonary artery are, as is well known, driven against the sides of their respective vessels, by the blood which is forced from the ventricles during the heart's systole, and expanded towards the ventricles, during the diastole, by the backward pressure of the blood, produced by the elasticity of the arteries.

The aortic valves occasionally lose their flexibility, in consequence of the development of excrescences, chalky concretions, etc., upon them, or of their abnormal union with each other, and thus become incapable of being pressed back against the walls of the aorta, and obstruct the entrance of the blood into the aorta. If the free borders of the valves are shortened or everted, or have foreign bodies attached to them, or if they be in part torn from their attachments, they are no longer in a condition to prevent

the reflux of the blood from the aorta into the left ventricle, during the heart's diastole.

We may readily ascertain, after death, whether the aortic valves duly performed their functions during life, by pouring water into the aorta; if the valves are healthy, the water does not flow back into the left ventricle, its reflux being prevented by closure of the aortic valves; but if the valves be defective, the water passes into the ventricle.

We cannot, however, apply this test to the mitral and tricuspid valves. If the apex of the left ventricle be opened, the aorta tied, and water then poured into the ventricle through the artificial opening, we shall find that the flow of the water through the mitral valves is occasionally prevented; but this does not invariably happen, and, by repetition of the experiment, we shall soon discover that no conclusions as to the actual condition of the valves can be drawn from it. If, again, one of the ventricles be filled with water, and its arterial opening closed, and pressure be then applied to the ventricle, the mitral or tricuspid valves (as the case may be) will be distended, but will not entirely prevent the reflux of blood, though in a perfectly normal condition. And the reason of this must evidently be sought for in the fact, that we are not able, after death, to imitate the contraction of the papillary muscles, and the general contraction of the heart's cavities. Whether, therefore, the mitral or tricuspid valves fully performed their functions during life, or not, is a question which we can only judge of, after death, from the conformation of the valves, of the tendinous

cords, and of the papillary muscles, and from the changes which the defect of these valves is wont to produce in the auricles.

Explanation of the Sounds heard in the Ventricles of the Heart.

By comparison of clinical observations with the facts revealed by post-mortem examinations, we find that a distinct first sound is very rarely heard over the left ventricle, when the mitral valves are incapable of preventing the reflux of the blood into the left auricle, during the heart's systole—*i. e.* when the mitral valves are defective. In such case, a murmur, synchronous with the systole, is generally heard over those parts of the thoracic walls against which the heart's apex beats; the first sound being distinctly audible over other parts of the præcordial region. The same is true of the right ventricle, when the tricuspid valves are defective; no distinct first sound is then heard over the right ventricle, but may still be recognized over the left ventricle, the aorta, and the pulmonary artery; in the right ventricle, it is generally replaced by a murmur.

The first sound in the ventricles, therefore, is for the most part produced by the sudden stoppage of the flow of blood towards the auricles, consequent upon the distension of the mitral and tricuspid valves, and thus, through the impulse of the blood, against these valves. Every blow creates a sound, and the sound is duller, the more yielding the nature of the body striking, or struck. The state of tension into which the valves are suddenly thrown by the

pressure of the blood, undoubtedly contributes to the production of the first sound, for cords and membranes always yield a sound when suddenly stretched, not solely in the air, as Gendrin and others have supposed, but also in water. The circumstance, moreover, that the first sound is often heard clear, with a clap, and at times even ringing, tends evidently to show, that the tension of the valves contributes to its formation.

The first sound is also produced by the impulse of the heart against the walls of the thorax. A blow struck with the finger, or with the apex of the heart firmly compressed, against the inner surface of the thoracic walls, produces a chinking (*klirrend*) sound, which differs in no particular from the ordinary first sound of the heart. If a part of the walls of the heart be somewhat separated from the thoracic walls during the ventricular diastole, but strike against them during the systole; or even if the heart beat against some other part of the thoracic walls during its systole than that beneath which it lay during the diastole, still, in either case, a chinking sound, or one exactly resembling the ordinary first sound of the heart, will be produced; for the heart's substance becomes firm during its systole. If the heart beat against that part of the thoracic walls, beneath which it lay during its diastole, the impulse will give rise to either a very dull sound, or none at all.

The muscular sound of the heart is always heard as a dull and prolonged sound, never as a clap; for no muscles ever yield a defined, clapping, or chinking sound; according to the division of sounds which

I have adopted, this muscular sound cannot be properly classed among the sounds of the heart, but must be rather looked upon as an indistinct murmur. Whether the contractions of the heart be really accompanied by such a murmur, I cannot say from observation on the living; but certainly cases are far from rare, in which the impulse of the heart is violent, and therefore its contractions strong, and in which nevertheless no first sound can be heard.

The causes of the first sound here adduced are not sufficient for the explanation of every case; and all attempts hitherto made to explain the different modifications of the first sound have proved signally unsuccessful.

The explanation of the causes of the second sound of the ventricles, presents still greater difficulties than that of the first sound; we cannot affirm that the second sound is always formed in the ventricles, when the condition of the heart is normal; for it seems probable, and sometimes indeed certain, that the second sound heard over the heart arises in the arteries, and, on account of its intensity, is propagated to a distance. But there are cases in which we are forced to admit that the second sound arises in the neighbourhood of the ventricles: cases, for instance, in which the second sound is barely audible over the base of the heart, but very loud and clear over its apex. A second sound of this description cannot be explained by the impulse of the heart against the thoracic walls, for there is no impulse during the ventricular diastole.

It is possible that the second sound may be oc-

casionally produced by the impulse of the blood against the walls of the ventricles during the diastole; such an impulse undoubtedly takes place in the left ventricle, when the aortic and mitral valves are defective. But only in one single case where there was deficiency of the aortic valves, have I observed the second sound to be louder at the heart's apex than elsewhere; in this case indeed it was remarkably strong and ringing. An increased second sound at the apex of the heart, is more frequently observed when the mitral valves are defective. When the left auriculo-ventricular opening is constricted, two muffled sounds, instead of a prolonged murmur, are occasionally heard over the left ventricle during the heart's diastole.

Gendrin availed himself of this fact to support his theory of the second sound of the heart: he considered that the double second sound was the consequence of the two ventricles not being filled synchronously: it appears to me much more probable, that these two sounds are merely divisions of a single murmur, which has its origin at the constricted opening. In fact, this murmur of constriction frequently becomes converted into two, or even three separate sounds, when the movements of the heart are weak. Moreover, in many cases, the murmur is distinctly heard at one spot, whilst over the parts around, two, or even three sounds—as though they were louder periods (*Momente*) of the same murmur—are audible.

Sounds Heard in the Arteries.

In all the larger arteries, a sound may be occa-

sionally heard in company with the pulse, having a tolerably close resemblance to an ordinary heart sound. I do not suppose that anyone can imagine, that sounds heard over the crural or brachial arteries can have had their origin in the heart, and been propagated from it; neither can the sounds heard over the carotid and subclavian arteries be considered as arising elsewhere than in the arteries themselves, in cases where no sounds can be recognized in the region of the heart, or only such as are weaker than those heard in the neck. This last fact may be frequently observed, and the only mode of accounting for the phenomenon is, to ascribe it to some especial conducting power in the parts which the sound traverses. It is an undoubted fact, that the propagation of sound through the thorax is affected by changes in the condition of its organs; but we frequently meet with cases in which the sounds are heard distinctly above or beneath the clavicles, while they are indistinct in the region of the heart, the lungs being in a perfectly normal condition; such cases are not explicable by the laws of conduction of sound. Bouillaud considers that the arterial sounds differ from the heart's sounds, having a resemblance to the sound produced by a fillip of the finger. The arteries at a distance from the heart most generally give rise to a simple toneless sound, such as Bouillaud describes; whilst those near the heart—the carotid, the subclavian, the pulmonary arteries, and the aorta—produce sounds for the most part as loud as those audible in the region of the heart; on the other hand, the sounds heard in the region of the heart are at times equally toneless.

The sound heard over the arteries synchronously with their pulsation, may be explained by the suddenly increased tension of their coats. The second sound is audible over the aorta and pulmonary artery, and also generally along the carotid and subclavian arteries. In other arteries we rarely hear any sound synchronous with the systole.

The second sound, heard over the aorta and pulmonary artery, is evidently produced by the shock arising from the regurgitation of the blood upon the semilunar valves, which takes place subsequent to the heart's systole. The blood which passes into the arteries during the systole, is subjected to compression by their elasticity, and when the forcing power of the heart ceases, is of necessity driven back towards the heart.

The reflux of the blood is suddenly arrested by the semilunar valves, and the impulse which they receive in consequence, is communicated to the walls of the vessels; the sound resulting is not only audible over the aorta and pulmonary artery, but is frequently communicated along the carotid and subclavian arteries, and this, too, even in cases where the coats of the aorta have lost the conditions necessary for the production of sound. The correctness of this explanation of the cause of the second sound, heard over the aorta and pulmonary artery, is placed beyond doubt by physiological and pathological observations; the sound does not appear to have any other cause for its production.

If its semilunar valves are defective, the second sound is not heard over the aorta, but the sound is

replaced by a murmur. The second sound of the pulmonary artery however remains distinctly audible. When the pulmonary artery is abnormally distended, which it always must be, when the pulmonary circulation is obstructed, its second sound becomes very loud, whilst the second aortic sound is either weak or inaudible, or is replaced by a murmur. The pulmonary artery, when thus distended, presses with increased force upon the blood within it, and consequently the backward stroke of the columns of blood against its semilunar valves becomes more forcible.

Dr. Rapp,—*Zeitschrift für rationelle Medizin*, vol. viii,—insists upon it, that the first sound arises only in the ventricles, the second only in the arteries. The first sound he considers, with Dr. Williams, to be a muscular sound; and the second, he attributes to the impulse of blood against the aortic and pulmonary valves. The explanation of the first sound, by impulse of the blood against the auricular valves, appears to him inadmissible, for the reason, that the valves are continually surrounded by blood; neither does he believe in the origin of a first sound in the arteries, and of a second in the ventricles, inasmuch as the phenomena are otherwise fully capable of explanation. Again, the interpretation of the second sound, heard over the ventricles, by a supposed tension of the tendinous cords of the auriculo-ventricular valves, is opposed by the character of the murmurs and sounds which are heard when the aortic valves are defective, and also by the fact, that the diastole does not follow instantane-

ously. Lastly, the explanation of the first sound in the arteries, by the tension of their coats, is in contradiction with the explanation of the first sound in the ventricles, by tension of the tendinous cords of the auricular valves; for the tension of the arterial coats cannot be less than that of the valvular cords, and consequently the first sound in the arteries should be as long and as loud as the first sound in the ventricles, which is contradicted by experience.

How far we are bound to admit the origin of the first sound in the arteries, and of the second in the ventricles, may be gathered from what we have already said; and on this score I have nothing further to add. I have already, in the second edition of my book, abandoned the explanation of the second sound of the ventricles by tension of the tendinous cords of the valves. The sound of the arteries during their diastole can hardly be otherwise explained than by strong tension of their coats; it is impossible to believe that the sound heard over the brachial or crural arteries could have been propagated from the first ventricular sound. Lastly, if the origin of the first sound in the ventricles is not to be explained by the impulse of the blood against the auricular valves, because these valves are continually surrounded by blood, then must the second sound of the arteries have some other cause than that to which it has been hitherto attributed; for the aortic and pulmonary valves are also continually surrounded by blood.

Variations in the Character of the Heart's Sounds.

The sounds of the heart and arteries vary in their

duration, strength, clearness, pureness, and pitch; they may be well defined, resembling the tic-tac of a watch, or prolonged, and murmuring. When the semilunar valves are healthy, the second sound of the aorta and pulmonary artery is well defined, particularly if the heart's action be strong, and closely resembles the flapping of a valve. The more defined, and consequently the more flapping, the character of the first sound over the ventricles, and the greater its resemblance to the second sound of the arteries, the more certainly may we conclude that it is produced by the impulse of the blood against the valves; but when the first sound is diffused over the ventricles, and ill-defined, it becomes doubtful whether it is formed in this manner; there is, in fact, another cause which may give rise to it. Such a sound, viz., one that is prolonged and heard over the ventricles, synchronously with the systole, I call an *indeterminate sound:* it gives no indication as to the condition of either the mitral or tricuspid valves, and cannot be classed among the true sounds or murmurs of the heart.

The sound heard with the systole over the ventricles, is often composed of a flapping and of a diffused and indeterminate sound. The flapping first sound of the heart is louder the greater the circumference of the valves and the finer their structure. The first sound over the ventricles loses its ringing character, when the free borders of the mitral or tricuspid valves are thickened; it becomes shorter than natural, resembling the sound produced by striking together two hard, not-ringing (*nicht-klin-*

yende) bodies. It may be very loud, and is frequently of a remarkably high pitch. The first sound over the ventricles may commence with a deep and end with a high tone, producing the sound of "twick" rather than of "tic": in some very rare cases, it is of so fine and ringing a character, as almost to resemble the sound caused by the sudden tension of a silk thread. It seems probable, that in such cases the tendinous cords alone produce the sound.

There is another variety of the first sound over the ventricles; the sound appears as though it were divided, and formed of two, or even three sounds, rapidly following upon each other, and blended together into a single one: the phenomenon appears to result from the circumstance of the valves not expanding instantaneously. The ventricular systole may also be accompanied by two distinctly different sounds. Gendrin considers this impossible, but Dr. Williams (on the other hand), in his *Lectures on the Diseases of the Chest*, admits the existence of a double first sound, and explains it by a want of synchronism in the contraction of the ventricles. I believe that in many cases this explanation is correct.

According to Augustus Baumgarten, the auricular valves are not closed by the systole of the ventricles, but by the contraction of the auricles preceding the systole, by which a still further quantity of blood is forced into the already distended ventricles, and their walls thereby rendered tense. Hamernjk has somewhat modified this opinion: he considers that the closing of the auricular valves is not always produced by the contraction of the auricles, but that it

is not unfrequently the consequence of the pressure to which the vena cava is subjected during expiration, without any assistance from the auricles. On this hypothesis Hamernjk explains the first sound in the ventricles, not by an impulse of the blood against the auricular valves during the systole, but merely by their increased tension; and the double first sound he attributes to a renewal of the tension produced by an undulation of the blood within the cavities of the heart. The closure of the valves of the aorta, and of the pulmonary artery, according to Hamernjk, precedes the contraction of these vessels, and consequently the second sound of the aorta and pulmonary artery is not coincident with the closure of their valves, but results from an increase of their tension when already closed; the double second sound is caused by a repetition of the increased tension consequent upon an undulation of the arterial columns of blood. In my opinion, the reflux of the blood into the vena cava, during the contraction of the right auricle, would not be prevented if the state of things suggested by Baumgarten really existed; if, under normal circumstances, the right auricle contracted in such wise as to force blood into the ventricles, pulsations of the veins of the neck would be invariably perceptible when the head was placed lower than the thorax, and the veins thereby rendered prominent. Now, as this is not the case, I cannot admit that the auricles normally contract so forcibly as to propel blood into the ventricles: I therefore consider that Hamernjk's views concerning the origin of the first single and first double

heart sound, and of the second arterial sound, are not well founded. I shall speak hereafter of his opinions concerning the motion of the blood in the veins of the neck.

The second sound over the ventricles has always a flapping character, but it may be nevertheless resonant, and somewhat prolonged; its continuity may be also broken and interrupted.

I have occasionally heard two sounds over the ventricles, in the place of the proper second sound: thus, instead of the ordinary "tic-tac," a "tic-tac-tac." These two sounds replacing the second sound, I once observed in a phthisical lad, a few days before his death; they continued audible till he died. Post-mortem examination did not bring to light any abnormal condition whatever of the heart. Such reduplication of the second sound is not explicable by any of the theories hitherto offered, of the cause of the second sound of the heart. When these two sounds are present, and the heart's movement slow, and the first sound, viz., that which corresponds with the ventricular systole, loud, they resemble the noise of a distant drumming. The existence of a double, and even triple second sound, in conjunction with constriction of the mitral orifice, has been already mentioned, and an explanation of the phenomenon been attempted.

The first sound over the pulmonary artery and the aorta, has generally but little of a clanging character, (*Klang*), and it has less, the thicker the coats of the arteries and the feebler the action of the heart.

The second sound in the pulmonary artery and aorta may also be broken; the interruption seems to arise from the circumstance of the semilunar valves not being all distended at the same moment.

In the ventricles the first sound is more prolonged than the second, in the aorta and pulmonary artery it is shorter, the accent falling on the second sound: the latter of these facts may be readily observed, when the sounds are loud. Hence, the measure of the sounds in the ventricles corresponds in some manner to a trochee (¯ ˘); and that of the sounds in the aorta and pulmonary artery to an iambus, (˘ ¯). When the accent over the ventricles falls upon the second sound, it is probable that this sound is not formed in the ventricles, but arises at the aortic and pulmonary valves, having been propagated thence by reason of its intensity.

The first and second sounds are sometimes of equal length over the ventricles, and over the arteries, the accent falling upon neither the one nor the other.

The interval between the second sound and a subsequent first sound is much longer than the interval between the first and second sounds. The latter interval is occasionally so short as to make the second sound over the ventricles appear like the accentuated termination of the first sound, and the first sound over the arteries as merely the commencement (*Vorschlag*) of the second sound. In other cases, however, the interval between the first and second sounds is nearly, or even quite as long, as that between the second sound and a subsequent first sound;

and this is particularly remarkable when the movements of the heart are slow. No particular signification can be attached to these accidental conditions of the sounds.

Laennec imagined that dilatation of the heart and thinning of its walls are indicated when the first sound over the ventricles is clear, resembling the second sound, and is audible over a considerable extent of the thorax; an opposite condition of the first sound, viz., when it is dull, weak, and scarcely audible, he looked upon as a sign of hypertrophy of the heart. We may examine a multitude of cases before we shall find one to corroborate such views.

THE MURMURS OF THE HEART.

The murmurs depending upon the heart's movements arise either within the cavities of the heart, or in the arteries or their coats, or within the pericardium.

Murmurs arising within the Cavity of the Heart.

These are designated as bellows, grating, sawing, filing, rasping, purring, whistling, and groaning murmurs. Laennec believed that such murmurs had no connexion with any structural changes of the heart, but were wholly the products of spasm; and his opinion was long the prevailing one. More extended experience, however, has demonstrated that in many cases organic changes of the heart's structure are associated with these murmurs, and changes of such a nature as to render their causes very explicable.

At the present time the general opinion is that murmurs within the ventricles are caused by friction of the blood against the walls of the heart, or against its valves. I would add, that murmurs may also be produced within the heart's cavities, by the rapid flow of a small stream of blood against blood that is quiescent, or moving less rapidly, or in an opposite direction to it. The fact of a murmur being created, when a small stream of any fluid, as water, blood, etc., is forced rapidly against a fluid at rest, may be demonstrated by direct experiment.

The following are the organic changes of the heart which give rise to murmurs within its cavities :—

1. Defect of the mitral, the tricuspid, and the aortic valves.

2. Constriction of the mitral orifice, or of the mouth of the aorta.

3. Irregularities—such as excrescences ; cartilaginous, calcareous, and bony concretions ; and blood-coagula—upon the endocardial membrane in the neighbourhood of the arterial openings, or upon the under surfaces of the aortic and pulmonary valves, or upon the auricular surfaces of the mitral and tricuspid valves. Excrescences, cartilaginous and calcareous concretions, blood-coagula, etc., attached to the lower half of the heart's ventricles, do not create murmurs, because the current of blood does not there possess the rapidity requisite for their production.

Those murmurs which cannot be directly attributed to organic changes of the heart, undoubtedly depend, for the most part, upon friction between the

blood and the walls of the heart. But how this friction produces the murmurs, has yet to be explained. A contraction of the heart, more forcible or rapid than ordinary, cannot of itself produce a murmur; and, on the contrary, murmurs may exist when the movements of the heart are slow.

The opinion that murmurs are caused by a particular condition of the blood, must be looked upon as hypothetical, until it has been shown in what that particular condition of the blood consists. It is not true that a watery state of the blood is a cause of murmurs in the heart; I have many times abstracted very watery blood from patients, in whom no murmurs existed. It is true that murmurs do occasionally arise in the heart after great loss of blood, and in anæmia, but not so constantly as to justify us in considering the anæmic condition of the blood as the only cause of the murmurs.

Andral believes that murmurs arise in the heart in cases of general plethora; he explains their presence, by supposing that the cavities are over-small, relatively to the quantity of blood which should pass through them in a given time. I have never observed a murmur that was thus produced, and cannot coincide with Andral's views; the passage of the blood through the heart's cavities, does not depend upon the blood, but upon the action of the heart.

Cardiac murmurs, unassociated with appreciable alteration of the heart's structure, are observed in the course of many different diseases. Chlorosis may be particularly instanced; but here, the murmurs are much more frequently heard in the arteries than

in the heart. The same remark applies to the faulty state of the blood produced by the cachexia of cancer.

A very loud blowing murmur has been sometimes heard, during the systole of the heart, in cases of acute rheumatism, where no change of the heart's structure could be found after death. During pregnancy, in puerperal diseases, at the onset of typhus, of small pox, and of severe inflammatory diseases, and under many other circumstances, a systolic murmur (either replacing the sound, or accompanying it) is occasionally heard in the heart or in the arteries.

Gendrin imagines that he can decide, from their timbre, whether murmurs depend upon any roughnesses within the endocardium, or are independent of any organic alterations of the heart: the latter kind, he tells us, resemble the sound of a bellows kindling a fire. I cannot admit this statement; it is vain to attempt to distinguish too particularly between the different kinds of murmurs; indeed, it matters little, as respects any conclusions which may be deduced from our observations, whether the murmurs are of a blowing, sawing, or rasping character.

We often find a bellows murmur suddenly converted into a sawing or rasping murmur, through accidental increase of the heart's action. Murmurs arising within the cavities of the heart, and the arterial murmurs, not only become louder when the heart's action is increased, but they also acquire a rougher, sharper, and more acute character; the reverse of this happens when the heart's action is weakened, the murmur becoming muffled and very indistinct, or disappearing altogether.

The character ascribed to a murmur is often very arbitrary; if several persons listen to the same murmur, one will probably describe it as a sawing, another as a filing, and a third as a buzzing murmur. Of much greater importance is it, that we should know where the murmur arises, whether in the left or the right ventricle, and whether it corresponds with the systole, or with the diastole of the heart;* for by these circumstances we judge of its signification.

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* Gendrin has attempted to mark with greater accuracy the sequence of the phenomena of the heart's action, in respect of the time of their occurrence. By *Présystole*, he represents the moment of time which immediately precedes the systole; the moment immediately following the systole, he calls *Périsystole:* in like manner, he describes a *Prédiastole*, a *Diastole*, and a *Péridiastole.*

A murmur arising at the root of the aorta, during the ventricular systole, is more périsystolic, than a murmur arising in the ventricles. A prédiastolic murmur is heard, when the waves of blood, in passing from the auricle into the ventricle, infringe upon roughened surfaces; should the roughness extend as far as the free borders of the valves, the murmur is prolonged into the second sound—*Percussion diastolique*—and ends with it, etc.

I cannot believe that anything is gained by such subdividing of the systole and the diastole. It is certainly true, that murmurs are more or less prolonged, that they immediately precede, or arise at the same time with, or follow closely upon, the heart's impulse; but if we recollect, that the duration of the sound does not depend wholly upon the continuation of the movement which originally occasioned it, and that the exact moment of the commencement of the systole, and of the diastole, is not marked, either by the heart's impulse, or by its sounds, we must consider the views of Gendrin, respecting the murmurs which precede and follow the systole and diastole, as hypothetical.

Murmurs arising in the Arteries.

Every kind of murmur which is heard within the left ventricle, may also be heard in the aorta. Murmurs arise in the aorta when its internal membrane is rendered uneven by excrescences, or cartilaginous and chalky concretions, the vessel itself either retaining its natural size, or being widened or contracted. Murmurs are produced at the aortic valves, when their under-surfaces are roughened; when foreign bodies occupy their free borders; when they have become rigid, or so united to each other that the blood flowing from the ventricle cannot force them back against the walls of the aorta; or when they are defective.

Murmurs are frequently heard over the subclavian and carotid arteries, not only when their internal membrane is roughened, but also when their coats are perfectly healthy.*

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* Hamernjk—*Untersuchungen über die Erscheinungen an Arterien und Venen*, etc., Prag. 1847—tells us, that no such thing as friction takes place between the blood and the walls of the arteries, because there is always a layer of blood lying motionless in contact with the walls. All arterial sounds are produced by vibrations of the coats of the arteries, and it is therefore improper to speak of murmurs in arteries; for this reason, Hamernjk calls them well-defined and ill-defined arterial sounds (*Töne*).

I have used the expression sound (*Ton*) as antithetical to murmur (*Geräusch*), in order to avoid the repetition of the terms, "normal, abnormal, limited," etc. If the adjectives. "well-defined and ill-defined" are employed, then, according to the ordinary use of language, the expression murmur (*Ge-*

When both ventricles are hypertrophied and dilated, and more particularly if the aortic valves are at the same time defective, a loud, rough, sawing, rasping, or groaning murmur is heard at each ventricular systole (and consequently with the pulsation of the arteries) over the subclavian and the carotid arteries, but less frequently over the abdominal aorta, the crural, brachial, and radial arteries. When the aortic valves are defective, a murmur may be occasionally heard in the carotid and subclavian arteries during the ventricular diastole; this phenomenon is most generally associated with aneurisms of the aorta.

A blowing systolic murmur is often heard over the carotid and subclavian arteries of perfectly healthy individuals, when their heart's action is increased, and particularly if their cervical muscles are at the same time put upon the stretch.*

The smaller the artery, the more rarely the murmurs just described are observed in it. Occasionally, when the smaller arteries are widened, we hear in them a continuous murmur, of a blowing, hum-

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räusch), is more correct than sound (*Ton*). The layer of blood at rest, in contact with the arterial walls, merely indicates that the current of blood is slower there, than in the middle of the artery; this is the case in all tubes, and doubtless also in the veins.

* Bouillaud tells us, that pressure with the stethoscope on every moderate-sized artery, produces a short, dull, blowing murmur; this I have frequently, but not invariably, observed; pressure, even against a strongly pulsating abdominal aorta, sometimes fails to produce a blowing murmur; and, on the other hand, in certain cases, the slightest pressure upon an artery, and even upon one as small as the radial, gives rise to it.

ming, hissing, whistling character, resembling the sounds produced by the air in a furnace, the murmur becoming stronger and higher in the ratio of the strength of the pulse; it is most frequently observed over the thyroid arteries, in cases of bronchocele; according to Professor von Kiwisch, it is also heard over the epigastric arteries of pregnant women, or when the abdomen is distended by ovarian or other tumours. We are also told by Kiwisch, that the so-called placental bruit does not arise in the vessels of the pregnant uterus, in the compressed arteries or veins of the pelvis, but in the epigastric artery; and the proof of this is, that the murmur disappears when the epigastric artery is compressed.*

When a moderately-sized artery communicates with a vein, we generally observe at the point of communication a very loud continuous murmur, the strength of which increases at each pulsation of the artery, and which is audible to a greater or less distance through the surrounding parts.

It is difficult to determine whether these arterial murmurs are caused by friction of the blood against the walls of the arteries, or by vibrations of the walls excited by their distension; the effect of a more than ordinary amount of friction, even when the arteries are healthy, must not be overlooked; and there is no reason why we should not admit that the arteries, through distension of their coats during

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* Von Kiwisch, *Beitrag zür Kenntniss der Anatomischen Beschaffenheit der Placenta, und Berichtigung der Ansichten über den Sitz des sogennanten Placentargeräusches*, Prag. 1849.

the systole, may produce a prolonged sound or murmur, instead of a short sound, the murmur becoming continuous whenever it lasts from one pulse to another.

In varicose aneurisms, the murmur is probably caused by the impulse of the stream of blood flowing from the arteries, against the blood in the veins.

Murmurs arising in the Pericardium.

A murmur is produced by friction of the internal surfaces of the pericardium, when rendered rough by the deposition of plastic exudation, or of tubercle, or of cartilaginous or chalky concretions; the murmur accompanies the movements of the heart,* and may be heard either during the systole or during the diastole only, or both during the systole and the diastole.

Laennec was not aware of the existence of this murmur, nor did he seem to pay any attention to it, even when Collin had shown that one of its varieties —the new-leather sound—was a sign of pericarditis.

Bouillaud describes three varieties of friction-sounds; viz., the rustling-sound, *bruit de frôlement;* the new-leather sound, *bruit de cuir neuf, bruit de tiraillement et de craquement;* and the grating-sound, *bruit de râclement.*

The first of these,—*bruit de frôlement,*—according to Bouillaud, bears a close resemblance to the fric-

* Gendrin will have it, that a pericardial murmur may be produced simply by increased action of the heart, especially in cases of palpitation, without there being any roughness of the pericardium. This I have never observed myself.

tion-sound of the pleura, and may be imitated by the crumpling of taffeta or parchment; it may accompany both the systole and diastole of the ventricles, but is heard loudest during the systole, and if the friction be considerable, becomes very like the rasping or sawing murmurs which accompany certain organic changes of the internal structure of the heart, and is only to be distinguished from them by being of a more superficial, diffused, and extended character.

A softer kind of friction-sound is heard when the opposed surfaces of the pericardium are dry and somewhat adhesive, but not covered, or only very partially, by false membranes, which is the case at the commencement of pericarditis. The superficial rasping or sawing-sounds indicate the formation of thick and uneven false membranes.

The new-leather sound is much more rarely observed than the rustling or rubbing-sound; indeed, it is only met with in those cases where the false membranes are thick, resistant, and elastic, and where perhaps they have formed partial adhesions, which are continually being put upon the stretch by the movements of the heart. The scratching-sound must be attributed to the presence of bony, or chalky, or fibro-cartilaginous concretions, or of points which, during the heart's movements, rub against each other, or some part of the pericardium.

There is no doubt that all these different friction-sounds of the pericardium have a real existence, and that many others might be added to them. According to my own experience, indeed, there is no kind of endocardial murmur, with the exception of the

whistling, which may not be imitated by a friction-sound of the pericardium; and no pericardial murmur which may not resemble an endocardial murmur.

In my opinion friction-sounds of the pericardium cannot be distinguished from internal murmurs of the heart, by being more superficial, more extended, and more diffused than these. A sound proceeding from a solid body, when strong and clear, appears to arise at its surface; but when of an opposite character, to come from some distant part of it. Thus, murmurs arising within the heart, may appear quite superficial, in consequence of their being inordinately loud, clear, high, and hissing, and may be audible over the whole region of the heart, and even beyond it; on the other hand, the friction-sound of the pericardium may be very weak and muffled, and thus appear to arise remotely. How are we to judge of the friction-sound which arises from the pericardium at the back of the heart?

I know no sign by which the friction-sounds of the pericardium can be distinguished from the internal murmurs of the heart, excepting this, that the internal murmurs correspond pretty exactly to the rhythm and to the natural sounds of the heart, whilst the pericardial friction-sounds seem to follow upon the movements of the heart. This distinctive sign is only available when the murmur is somewhat prolonged; if it be of short duration, we cannot determine whether it is endocardial or pericardial.

I do not believe that the pericardium can produce a friction-sound, so long as its surface is smooth or uncovered by plastic exudation; at least, I have

never seen such a case. The false membranes may be very thick, firm, uneven, and elastic, and may have formed adhesions, and still produce no other sound than a slight rubbing or rustling, having the character of a bellows-murmur. Again, scarcely any friction-sound may be heard, in consequence of the feebleness of the movements of the heart, although the pericardium be completely covered by rough, thick, false membranes, and the amount of serous effusion small. Hence the strength of the friction-sound and its roughness depend upon the force of the heart's movements, as well as upon the nature of the false membrane.

A friction-sound accompanying the movements of the heart, does not invariably arise from the inner surface of the pericardium; it may depend also upon a roughened condition of that portion of the pleura which covers the unattached parts of the pericardium. The sound is produced by the rubbing of the pleura which covers this free portion of the pericardium, either against the thoracic walls, or against the surface of the lungs: being caused by the action of the heart, it coincides with its movements as completely as though it had been produced within the pericardium. The murmur thus arising external to the pericardium, exactly resembles the murmur arising within it. I once thought, that a distinction between them might be found in the circumstance of the external murmur being alternately increased and diminished in force during the respiration; but it appears that the internal pericardial murmur is likewise frequently affected in a similar way.

Murmurs arising in the Jugular Veins.

In a great many young persons a continuous murmur, which is apparently increased in strength at each systole of the heart, is audible in the hollow between the heads of the sterno-cleido-mastoideus muscles, particularly on the right side of the neck: this murmur was supposed to have had its origin in the arteries of the neck, until Dr. Ogier Ward, in 1837, and Hope and Aran after him, demonstrated that it arose in the jugular veins. Bouillaud called this sound *bruit de diable*, in consequence of its having a resemblance to the humming of a top (Top-murmur, Nun's-murmur). Laennec pointed out a variety of it, under the name of musical arterial bruit. This bruit de diable, heard at the part above referred to, disappears when the current of blood is interrupted by pressure upon the internal jugular vein; it also disappears upon a deep inspiration, by which the blood is driven back into the jugular veins; and lastly, in every position of the body where the head lies lower than the thorax, and the flow of blood into the jugular veins impeded. The murmur is loudest in the erect position and during inspiration, but is never heard when the jugular veins are distended with blood.

The increase of the murmur at each systole of the heart, is only apparent, and is occasioned by the sounds and murmurs which proceed from the carotid and subclavian arteries, and which are heard in the neck during the systole. Deep expiration, as well as a low position of the head, both of which cause

the venous murmurs to disappear, have no effect in arresting arterial murmurs; on the contrary, such murmurs are not unfrequently thereby produced, in cases where healthy sounds only are heard, so long as the respiration is tranquil, and the head erect.

Although, from these facts, there appears to be no doubt that the bruit de diable has its origin in the jugular veins, its mode of production is not very clear. Hamernjk explains the murmur in this way: the less the quantity of blood present in the vena cava, the more rapid will be the flow of blood through the jugular veins during inspiration, and the smaller the current of blood. Now, the internal jugular vein is so attached at its lower part, as of necessity always to retain a certain width: the diminished stream of blood can only fill this wide space, by passing through it with an eddying movement; this movement is communicated to the walls of the vein and the parts around, and is rendered sensible to the touch by vibrations, and to the ear by murmurs. It is well known, that the bruit de diable is only heard in the veins of the neck, and more frequently on the right side than on the left. This fact, in Hamernjk's opinion, confirms his views; for the conditions which. according to him, are necessary for the production of the bruit, exist only in the internal jugular veins. and in a much more perfect manner in the right than in the left. The bruit has hitherto been considered a sign both of anæmia and of spanæmia; Hamernjk looks upon it as a sign of anæmia only. I have for many years looked upon it as a sign, neither of a watery condition of the blood, nor of deficiency of

blood; for it exists even in young and healthy individuals; in such persons, the vena cava is never so distended with blood as to cause its regurgitation into, and swelling of, the jugular veins. Moreover, the jugular veins are shorter in young than in old persons, so that they may be stretched by certain positions of the head; this last circumstance may explain the absence of the bruit in old age, when the vena cava contains but little blood.

Rules for the diagnosis and determination of the sounds and murmurs of the Heart, the Pericardium, the Aorta, and Pulmonary Artery.

When an observer auscultates the præcordial region, and the points of the thorax which correspond to the course of the aorta and pulmonary artery, he will everywhere hear, either the ordinary tic-tac, and no murmur synchronous with the heart's movements; or he will hear the tic-tac at certain points only, but still no murmur; or, again, he will nowhere hear the tic-tac, or only at some special points, but in its place, at one, or several, or at every part, one or more murmurs.

The sounds, as well as the murmurs, are always loudest and most distinct at those parts of the thorax which are nearest to their points of origin—exception being made of those cases, in which the sound or murmur is either increased by resonance, or muffled by the accidental intervention of a bad conductor of sound. The heart's sounds and murmurs are increased by resonance in cases of pneumothorax, and when they arise in the neighbourhood of empty cavi-

ties; but they are rendered duller by the intervention of lung, or of exudations, between the thoracic walls and the heart.

In accordance, therefore, with what has been here stated, we ascribe to the left ventricle the origin of all those sounds and murmurs which are heard most distinctly at that part of the thoracic walls against which the heart's apex strikes; and to the right ventricle, those which are most clearly heard over it—that is, over the lower part of the sternum. Those sounds and murmurs, again, which are heard loudest along the course of the ascending aorta, *i. e.* a little to the right of the centre of the sternum, and from thence upwards, arise in the aorta; the sounds heard most distinctly along the course of the pulmonary artery—somewhat to the left of the centre of the sternum—are formed in the pulmonary artery. It must not, however, be forgotten, that pericardial murmurs may present themselves at every part of the thorax here referred to.

Of the position of these different parts, that of the left ventricle of the heart is, in the greater number of cases, the most readily and most certainly determined. The point furthest to the left, where the impulse of the heart can be felt—not the mere concussion thereby produced, for this may extend to some distance from the point of impulse—may be taken as a fixed point, for the determination of the position of the other parts; this point will not fail to represent the position of the left ventricle, except in the case where there is very great dilatation of the right, in conjunction with diminution of the left ventricle.

The ascending aorta always lies somewhat to the right of the vertebral column, consequently its sounds and murmurs must always be sought for over the middle, and somewhat to the right of the sternum. The base of the heart—and consequently the aortic and pulmonary valves—are almost invariably situated behind the middle of the sternum, and are only found lower down, in cases of dilatation and hypertrophy of the heart. The position of the right ventricle is variable, and cannot be determined, unless that of the left ventricle and aorta has been previously ascertained; it yields no special indication. The same remark is true of the position of the pulmonary artery.

It is of great importance for us to be able to determine, whether the murmurs heard arise in the heart and arteries, or in the pericardium; for the murmurs which take their origin in the pericardium, may be heard over the same parts as those which arise in the heart and arteries.

If the duration of the murmur be so short, or the heart's movements so indistinct to the touch, or so irregular, as to prevent our deciding the question by the criteria above given, still we frequently may, by a comparison of all the signs present, diagnose with pretty considerable accuracy, if not with actual certainty, the seat of origin of any particular murmur. Thus, murmurs arising within the heart and arteries, are occasioned by changes in the state of their internal membranes, and particularly of their valves; which changes, by their influence on the circulation, and through the effects which they produce in the

heart and the aorta, are generally rendered manifest by other signs, as well as by murmurs. On the other hand, the exudations which give rise to pericardial murmurs, are very often associated with serous effusion, in such quantity that its presence may be determined by percussion.

When we have decided that the pericardium is the source of the murmur heard, we must next endeavour to ascertain, whether it arises on its internal or external surface, *i. e.* whether the morbid condition occasioning it exists within or without the pericardium. As we have already said, the character of the murmur itself does not aid our diagnosis; we must therefore take into consideration the percussion signs, the position of the heart, and the exact part where the murmur is heard. If percussion indicates the presence of effusion in the region of the heart, and if the heart be not thereby displaced, we may conclude that the effusion is in the pericardium; and it then becomes very probable, that the murmur also proceeds from the internal surface of the membrane. But if again we determine the existence of effusion in the precordial region, and at the same time find the heart forced from its natural position, we then conclude that the effusion is external to the pericardium,—that is, in the pleura,—and the murmur, in all probability, arises upon the outer surface of the pericardium. If percussion do not indicate effusion, and the murmur be heard loudest at the middle line of the sternum, it arises within the pericardium: but if it be heard loudest at the periphery of the sternum, or beyond this, then it becomes very doubtful, whe-

ther the morbid condition exists within or without the pericardium.

When we have determined that the murmur arises within the heart or the arteries, and have also ascertained at what points of the thorax it is heard loudest, it still remains for us to decide—that is, in case the murmur is audible over a considerable extent of surface—whether it has one or several sources. To ascertain this, we must make a comparative examination of the other signs present. We have already observed, that changes of the inner coverings of the heart and aorta, and particularly of their valves, give rise to murmurs; such changes also produce disturbance in the general circulation, and disturbances of the circulation bring forth a variety of phenomena, and amongst others very frequently abnormities in the form, the size, and the nutrition of the heart, which again present their own especial indications: thus we are enabled by one set of signs to correct another, and by their comparison to arrive at right conclusions.

When we have satisfactorily determined the point of origin of the sound or the murmur, it remains for us to ascertain whether the sound or murmur be synchronous with the heart's systole or diastole; this may generally be at once decided from the rhythm of the sounds or murmurs; for the interval between the second sound or murmur and a subsequent first sound or murmur, is, as a rule, longer than the interval between the first and the second sound or murmur. A person, however, who is not much practised in auscultation, will do well to feel for the heart's impulse

with his fingers while listening with his ear. The sound or murmur synchronous with the heart's impulse, will be found to correspond with the heart's systole—the first sound; the sound or murmur heard subsequent to the heart's impulse, to be synchronous with the beginning of the diastole—the second sound. This combination of auscultation with palpation of the heart's impulse, is particularly necessary in the investigation of the sounds or murmurs of the aorta and pulmonary artery. Indeed, it frequently happens that the irregularity of the heart's movements is such, that even the most skilful auscultators cannot decide, from the rhythm of the sounds and murmurs, which is the first and which the second; he must, in such cases, of necessity consult the heart's impulse; and the same remark is true when only one sound or one murmur is audible.

Signification of the sounds and murmurs heard over the Ventricles, the Aorta, and Pulmonary Artery.

In the left ventricle, during its systole, a sound without murmur—first sound—indicates that the mitral valves duly perform their office, and that there is no reflux of blood from the left ventricle into the left auricle.

A murmur only—first murmur—indicates either imperfect closure of the mitral valves during the systole, and friction of the blood against some roughened parts of the valves through the reflux of the blood into the left auricle; or, that a rapid current of blood from the left ventricle is forced in an opposite direction to the blood flowing from the left

auricle. The murmur may also arise through friction of the blood against irregularities situated upon the surface of the internal membrane of the left ventricle, about the arterial opening, which do not interfere with the closure of the mitral valves; or it may be produced by a combination of the first and second of these causes.

When the closure of the mitral valves is not complete, a portion of blood at each systole regurgitates from the left ventricle into the left auricle, causing distension of the left ventricle and of the pulmonary veins and arteries, so that increased efforts of the right ventricle become necessary, in order to force the blood onwards through the distended vessels; the pulmonary artery, thus strongly dilated, presses with an exaggerated force upon the blood within it, and drives it more suddenly and more forcibly backwards against the semilunar valves during the heart's diastole, whereby the second sound of the pulmonary artery is increased.

We must not conclude, from the fact of a murmur being heard in the left ventricle during the heart's systole, that the mitral valves are defective, unless we at the same time ascertain that there is an increase in the intensity of the second sound of the pulmonary artery: if the force of this sound be not increased, the murmur then indicates that the surfaces of the valves, or the lining-membrane of the ventricle, about the arterial openings, are roughened. It is at these parts that the stream of blood possesses such velocity, that its friction can create a murmur;

roughnesses about the apex or the middle of the ventricle, produce no murmur.

An increase of the second sound of the pulmonary artery almost invariably accompanies defect of the mitral valves; I believe that it can only fail to do so when the coats of the pulmonary artery have lost their elasticity, and do not contract rapidly after distension.

When the lining-membrane of the left ventricle is inflamed, — *endocarditis*, — a systolic-murmur will arise in that ventricle, provided there be any defect of the mitral valves, produced either by tumefaction of their free borders, by lengthening or shortening of their tendinous cords, by excrescences on the valves or their tendons, or by deposition of fibrinous matters upon them, or by any roughness of surface about the ostium arteriosum. Hence a systolic murmur heard over the left ventricle, does not indicate endocarditis, unless when associated with such disturbance of the organic functions as usually attends endocarditis, and when the murmur did not exist previous to the functional disturbance.

A sound associated with a murmur has the same signification as a murmur without a sound. For example; the sound may result from the perfect closure of the valves, and the murmur from roughness of the ventricular surface about the ostium arteriosum: or the sound may arise from distension of some of the pouches of the mitral valves which still retain their normal condition, whilst their perfect closure is prevented by the abnormal condition of the remaining portion of their borders, and a mur-

mur thus produced; or the sound may proceed from the heart's impulse, and the murmur from defect of the mitral valves.

Failure of both sound and murmur is a phenomenon of no value as indicative of the condition of the mitral valves. When we meet with such a case, we must endeavour to decide as to the closure of the valves, by a careful consideration of all the other coexisting phenomena. The sound may fail, although the closure of the valves is perfect, and the reflux of blood prevented, whenever those conditions which render the sound dull are present in a high degree.

The murmur may likewise fail, though there is deficiency of the mitral valves, and though the blood in its reflux impinges upon no roughened surface nor flows with any great velocity. Failure of the murmur with defect of the valves, is of rarer occurrence than failure of the sound in connexion with their perfect closure. If neither sound nor murmur can be heard over the left ventricle during its systole, we must turn our attention to the state of the second sound of the pulmonary artery, and ascertain whether there is any diastolic murmur audible over the left ventricle; if the second sound of the pulmonary artery is increased in force, and the second sound over the left ventricle replaced by a murmur, it becomes more than probable that the closure of the mitral valves is not perfect, the phenomena present indicating contraction of the mitral valves, which very rarely happens without their being also defective.

Although the second sound of the pulmonary

artery is increased, and both sound and murmur absent in the left ventricle, during the heart's systole, and the second sound in the left ventricle, either unaccompanied by a murmur or inaudible, still we are not justified in considering the mitral valves defective, unless we can show that no other cause is present to account for hypertrophy of the right ventricle, except that of the deficiency of the valves in question. The other more particular causes of hypertrophy of the right ventricle are, curvatures of the spine, extensive and long-standing thoracic effusions without marasmus, etc.

A sound (*Schall*) may be sometimes heard in the left ventricle during its systole, of such an indefinite character, that it cannot be classed either as a sound or a murmur. For the determination of the condition of the mitral valves when such an indefinite sound is heard, the same rules must be applied as are laid down in the case where there is failure of both sound and murmur.

In the left ventricle during its diastole, a sound unaccompanied by a murmur—second sound—indicates that there is no constriction of the left auriculo-ventricular opening, and that the blood, in passing from the left auricle into the left ventricle, does not flow over any roughened surface.

A murmur accompanied by a sound, or a murmur alone, indicates either constriction of the mitral orifice, with roughness of the narrowed surface of the canal, or the presence of rough eminences upon the auricular surface of the mitral valves, unaccompanied by constriction of the opening. Constriction of the

mitral orifice causes the blood to accumulate in the left auricle, and in the pulmonary veins and arteries, producing hypertrophy with dilatation of the right ventricle, and an increased second sound of the pulmonary artery, much more readily than mere deficiency of the mitral valves. The increased second sound is not observed when roughnesses alone exist on the auricular surface of the valves, without constriction of the auriculo-ventricular opening, provided it be not accidentally present through some other cause.

The more constricted the mitral office is, the longer will be the time necessary for the flow of the blood into the ventricle, and the more prolonged and louder the murmur. It is in cases of this kind especially, that vibrations are felt when the hand is laid upon the precordial region,—a phenomenon likened by Laennec to the purring of a cat—*frémissement cataire.*

These vibrations are indeed often visible, and the murmur then resembles the humming (*Summen*) of a distant bell. The murmur resulting from constriction of the mitral orifice, may be so prolonged as to be only momentarily interrupted during the systole, particularly if the heart's movements be rapid: an unpractised observer will find it difficult, in such a case, to decide by the ear whether the murmur occurs during the systole or the diastole; he will therefore do well to aid his judgment by palpation, and by ascertaining the presence or not of the "purring" during the diastole.

Failure both of sound and murmur has no distinct

signification; particular regard must therefore be paid to the nature of the second sound of the pulmonary artery, and to the first sound or murmur, and, above all, to the action of the mitral valves during the ventricular systole, if we wish to obtain a satisfactory idea of their condition during the diastole. If the first sound is audible in the left ventricle, unaccompanied by a murmur, and the second sound of the pulmonary artery is not increased, we have no grounds for concluding that the mitral valves are abnormal; but if a systolic murmur be heard over the left ventricle, and the second sound of the pulmonary artery be increased, then failure of the second sound in the left ventricle must be attributed to defect of the mitral valve.

The presence of a systolic murmur in the left ventricle, with absence of sound and murmur during the diastole, and increase of the second sound of the pulmonary artery, are signs which in most cases indicate defect of the mitral valves, without constriction of the mitral orifice; at the same time, they do not preclude the existence of such constriction. If the left auriculo-ventricular opening be constricted (and this condition is almost always associated with defect of the mitral valves), the diastolic murmur is generally loud and prolonged, the systolic being weak, and of short duration, or even inaudible; but there are exceptions to this, for the systolic murmur may be loud and prolonged, while the diastolic is weak and short, or inaudible. These differences most probably depend upon the form and direction of the constricted canal, and upon the situation of its roughened surfaces.

Lastly, when the movements of the heart are rapid, the systolic may be so confounded with the diastolic murmur, that only one prolonged murmur is heard, commencing with the systole, continuing through the first part of the diastole, and broken only by the very short interval which corresponds to the quiescent state of the ventricle. This combined double murmur is in no way distinguishable from a prolonged single murmur; and it is only when the heart's movements are slow, that it becomes resolved into two distinct murmurs.

When there is an absence both of sound and murmur over the left ventricle during the systole and the diastole, we cannot affirm the existence of any abnormal condition, such as defect or constriction of the mitral valves, by which functional derangement of the heart is produced, unless the second sound of the pulmonary artery is distinctly increased, and its increase not attributable to any other cause.

An indeterminate sound, which cannot be designated either as a sound or a murmur, may be sometimes heard in the left ventricle, during both the systole and diastole of the heart. In such case, we must draw the same conclusions as when neither sound nor murmur are audible.

In the right ventricle, during the systole, a sound without a murmur—first sound—indicates perfect closure of the tricuspid valves, and, consequently, that there is no reflux of the blood from the right ventricle into the right auricle during the ventricular systole.

A murmur alone, or a sound accompanied by a

murmur, indicates either imperfect closure of the tricuspid valves, with roughness of their free borders, or that there are roughened spots upon the conus arteriosus, the valves duly performing their office; this latter case, however, rarely happens. When the closure of the valves is perfect, the systolic murmur heard, arises more frequently from roughnesses upon the tendinous cords, which are inserted into the septum of the ventricles near to the conus arteriosus, than from roughnesses upon the surface of the conus arteriosus itself.

Defect of the tricuspid valves gives rise to accumulation of blood in the right auricle, vena cava, etc.; hence results swelling of the veins of the neck, which would otherwise be retracted and shrunken, so long, at least, as the head and neck are placed higher than the thorax, the abdomen, and the lower extremities. The blood driven back into the right auricle, during each systole of the right ventricle, causes an accumulation of blood in the venæ cavæ; the jugular veins become full and distended, and, at each diastole, seem to retract and collapse; this phenomenon represents the so-called pulsation of the jugular veins; there is generally, however, no pulsation perceptible to the touch. The undulations of the blood may be felt when the distension of the veins is considerable, and particularly in the subclavian vein, when it projects above the clavicle.

Deficiency of the tricuspid valves will therefore be recognized by systolic murmur in the right ventricle, with synchronous pulsation of the jugular veins. Such a murmur, however, unaccompanied by pulsa-

tion or distension of the veins, does not indicate defect of the tricuspid valves. When the murmur exists, and the cervical veins are distended, but do not pulsate, the tricuspid valves may be defective; but in such case, either the heart's action is feeble, or the deficiency of the valves is very slight, so that but little blood is driven backwards at each systole.

I will here state my objections to Hamernjk's opinions, concerning the use of the valves in the veins of the neck, and the movement of the blood in the venæ cavæ. The most essential part of what here follows, I have taught ever since 1836.

The object of the valves of the veins is not to aid the flow of blood towards the heart, or to lessen the weight of the columns of blood, but solely to limit to the larger veins the unavoidable reflux of the blood produced by movement of the body, or by pressure upon any single parts, and thereby to prevent injury of the capillary vessels. Thus, for example, rubbing the arm down and pressing it at the same time, would cause rupture of the capillaries, but for the valves of the veins; in fact, but for them, any sudden contraction of large muscles would scarcely fail to produce effusion of blood.

The valves of the veins of the neck have no other object than this; most assuredly, they have not the function attributed to them by Hamernjk, viz.: to serve as fixed points for the columns of blood in the venæ cavæ, during ordinary expiration, so as to render possible a forcing of blood into the right ventricle, by the movements of expiration.

Healthy lung-tissue possesses a contractile power,

which occasions the arching upwards of the diaphragm, and the sinking-in of the intercostal spaces; and the power does not cease with expiration. It may be shown, by experiments on animals whose lungs are healthy, that when the thorax is opened, the lung frequently contracts into less than half the space it occupied towards the end of expiration, when the thorax was uninjured. The continuance of the contractile power of the human lung during expiration, and to the end of expiration, is proved by the fact that, in healthy persons, the percussion-sound is as little tympanitic during expiration as during inspiration; whilst the loss of contractile power of the lung in disease, is rendered manifest by the presence of a tympanitic sound during inspiration and expiration—a diminution of its contractile power is shown by the presence of the tympanitic sound during expiration only. Consequently the healthy lung exercises an influence over the walls of the thorax and the contents of the thoracic cavity, both during inspiration and expiration, and this influence is greater during inspiration than during expiration; the difference, however, can hardly be considered sufficient to account for any perceptible widening or narrowing of the venæ cavæ during the respiratory movements, for the tension of the intercostal spaces does not visibly remit during tranquil expiration.

When the respiration is tranquil, no pressure is exerted on the vena cava or on any of the thoracic organs during expiration, and consequently the blood in the vena cava is not pressed upon, nor the valves of the veins of the neck called into action; neither

need we assume that the liver is displaced in some most extraordinary and original manner, in order to serve as a valve for the inferior vena cava.

Pressure of the thoracic walls upon the lungs, and through the lungs upon the contents of the thorax, takes place only during expiration, when the contractile power of the lung is insufficient to expel the air it contains. In such cases, the blood is driven back from the vena cava into the veins of the neck and of the extremities; and, were it not for the presence of their valves, the vena cava would be completely emptied of blood whenever a certain degree of pressure was exerted upon it by the lungs. There is no necessity for a valve in the vena cava between the thorax and the abdomen, because, at each forcible expiration, the organs of the abdomen undergo the same pressure as the lungs, and the blood in the inferior vena cava and in the hepatic veins, is as much pressed upon as the blood in the superior vena cava.

It is hardly necessary to refer to the fact, that the blood is not forced from the venæ cavæ into the auricle or the ventricle of the heart, during either ordinary or forced expiration. The contractile power of the lungs, when the expiration is tranquil, and the pressure of the thoracic walls, when it is forcible, acts with exactly the same power upon the heart as upon the venæ cavæ. It is not my intention to pass in review the different theories of Hamernjk, which result from his views concerning the influence of expiration upon the circulation of the blood. I will merely add a few remarks on the venous pulse.

A reflux of the blood into the venæ cavæ during

forcible expiration, is produced by the pressure of the walls of the thorax and abdomen, by contraction of the right auricle, and by contraction of the right ventricle when the tricuspid valves are defective.

This reflux of the blood into the vena cava, becomes manifest in the cervical veins, either when the blood is driven back into them, or when it is impeded in its onward movement; the first case happens when the valves of the cervical veins are defective, the second, when they duly perform their office. So long as the valves act, the veins have only to sustain the pressure of the blood behind, and are consequently but slightly distended; but if they do not close, they have to bear a share of the pressure which is exerted upon the thoracic and abdominal organs during forcible expiration, and are consequently much distended.

When expiration is performed by the contractile power of the lungs alone, either very slight or no undulation whatever is observed in the cervical veins during the respiratory movements; and this is true, even though their valves do not close. The widening of the vena cava (although very inconsiderable) which takes place within the thorax during inspiration, in consequence of the increased pressure exerted upon the inferior vena cava by the contraction of the abdominal muscles, is compensated by its contraction within the thorax during expiration, consequent upon the enlargement of the abdominal cavity and diminished pressure upon the inferior vena cava. When the contractile power of the lungs is of itself insufficient to expel the air, the cervical veins, if

their valves are defective, become distended, the blood being forced back from the vena cava into them during expiration; if the valves close, and the blood be retained in the vena cava, the cervical veins will also be distended as suddenly, but not as forcibly.

During inspiration, the swelling of the cervical veins diminishes or disappears. This rising and sinking of them during the respiratory movements, does not represent what it called the venous pulse.

Contraction of the right auricle, the object of which is to propel the blood into the ventricle, must also cause a reflux of blood in the vena cava; and this reflux becomes visible in the cervical veins, even if their valves close, whenever the blood in them presses upon the blood in the vena cava, or the blood in the vena cava upon that in the cervical veins. In such cases, the impulse imparted to the valves by the reflux of the blood, is necessarily communicated to the columns of blood above the valves; the undulations thus produced are synchronous with the increase of the blood-columns, which occurs during the closure of the valves, and is occasioned by the unceasing reflux of the blood.

In health, the vena cava is never completely filled with blood, and consequently the blood in the cervical veins does not press upon that in the vena cava, so long as the head is higher than the thorax; in such a position moderate reflux of the blood might occur, without manifesting itself in the cervical veins; but if the head be placed lower than the thorax, the

cervical veins at once swell out, because the blood in the vena cava now rests upon that contained within the veins, and every reflux of blood from the vena cava becomes manifest as an undulation in the cervical veins.

In healthy persons no such undulatory movement of these veins is visible, even when they are considerably swollen and the head is placed low; the reason of this is, that, under normal circumstances, the right auricle does not contract so as to communicate movement to the blood. In abnormal states, however, and particularly when much distended through obstruction to the pulmonary circulation, the right auricle contracts vigorously, and its contractions produce pulsations in the cervical veins. These contractions occur either in the course of the ventricular diastole, or they somewhat precede the ventricular systole: two, or even three contractions, may be associated with a single systole.

When the tricuspid valves are defective, regurgitation of blood into the vena cava takes place at each ventricular systole; the undulation thereby created reaches the blood in the cervical veins, even though their valves be closed, and produces a pulsation there, provided the blood in the cervical veins rests upon the blood in the vena cava.

Experience teaches us, that contractions of the right auricle may be synchronous with the ventricular systole; pulsations therefore of the cervical veins cannot be considered as indicative of defect of the tricuspid valves, unless associated with a systolic murmur of the right ventricle. Pulsation of the

cervical veins, caused by contraction of the right auricle, does not continue for any length of time synchronous with the ventricular systole; several venous pulsations may correspond to a single systole; and an undulatory movement of the cervical veins may be produced solely by violent movements of the heart, independent of any contractions of the right auricle or defect of the tricuspid valves.

The absence of sound and murmur, or the presence of a sound of such indefinite character, that it cannot be classed either as a sound or murmur, does not offer any distinct indications. We must, in such cases, in order to obtain some probable idea of the condition of the tricuspid valves, proceed in the manner recommended for ascertaining the condition of the mitral valves, in the case where there is absence of sound and murmur in the left ventricle during its systole—with this difference, that in the one case regard must be had to the force of the second sound of the pulmonary artery, and, in the other, to the condition of the jugular veins.

I have never observed *a diastolic murmur over the right ventricle.* Contraction of the right auriculo-ventricular opening is of very rare occurrence. The signification of the second sound in the right ventricle, as well as its absence, may be gathered from what has gone before.

A sound heard in the aorta during the heart's systole, unaccompanied by a murmur,—first sound, — does not necessarily indicate that the aorta is in a perfectly normal condition; it is heard loudest during the violent action of an enlarged heart,

when the coats of the aorta are healthy, and its width adapted to the size of the heart. The sound is dull when the coats of the aorta are thicker and less elastic than natural; when the heart's action is weak; and when the diameter of the aorta is either too large or too small in comparison with the size of the heart.

A murmur alone, or a sound accompanied by a murmur, indicates the presence of roughened patches upon the inner surface of the aorta, or upon the under surface of the semilunar valves. But a murmur of a dull character may arise in the aorta, independent of such abnormal conditions.

The absence of sound and murmur, or a sound of such indeterminate character as not to be capable of being classed either as a sound or murmur, is produced by an exaggeration of the same causes that give rise to dulness of the first aortic sound.

A sound heard in the aorta during the ventricular diastole, unaccompanied by a murmur, — second sound,—indicates closure of the aortic valves. It is strong and loud, when the coats of the aorta and the aortic valves are healthy, and the action of the heart strong; but the sound becomes dull, when the aortic valves and the coats of the aorta are thicker and less elastic than natural, when the heart's action is weak, and when the mitral valves are defective, or the mitral orifice constricted. In some rare cases, the second sound of the aorta has a ringing character (*Klang*), and then really becomes a musical sound (*Ton*). This ringing second sound is not heard, except in old people. In one case, where it was very

marked, the ascending and transverse aorta, as well as the large arteries given off from it, were found, after death, converted into nearly solid canals by the deposition of chalky concretions, the aortic valves being healthy.

A murmur without a sound, when it is prolonged, and heard above the base of the heart, indicates defect of the aortic valves, and the presence of roughnesses upon their free borders. If the murmur is short, and only heard high up over the aorta, it is caused by the presence of roughnesses upon the inner surface of the aorta; we cannot, therefore, by consideration of the murmur alone, decide whether the closure of the aortic valves is complete.

A murmur terminating in a sound—a murmur bounded by a sound—depends upon the presence of roughened patches upon the inner surface of the aorta, when the closure of its valves is perfect. The columns of blood driven against the valves during the diastole, produce a murmur, through friction against the roughened patches on the inner surface of the aorta; but the murmur continues only so long as the current of blood is in motion, and therefore ceases with the closure of the valves, which closure produces a sound.

A murmur accompanied by a sound, the murmur being of longer duration than the sound. The aortic valves, in such case, are distended by the blood, but are incapable of performing their office; the blood regurgitates into the left ventricle, and causes a prolonged murmur.

The absence of both sound and murmur, or a

sound so dull that it cannot be characterized either as a true sound or a murmur, has no distinct signification. When such indefinite signs are present, we may perhaps arrive at some idea of the condition of the aortic valves, by taking into consideration the consequences of their deficiency. When the aortic valves are defective, the power by which the blood is driven onwards by the arteries, acts backwards upon the left ventricle during its diastole, causing the heart to become dilated and hypertrophied. If, therefore, we discover signs of hypertrophy of the left ventricle, and the signs of closure of the aortic valves are doubtful, it is most probable that these valves are not perfect; but if the size of the left ventricle be normal, then we may be certain that the closure of the valves is complete, even though the second aortic sound should be very indistinct, or altogether absent.

Hypertrophy with dilatation of the right ventricle, caused by defect of the mitral valves, is occasionally accompanied by a systolic murmur in the pulmonary artery; this murmur perhaps depends upon some softening of the inner membrane of the distended artery. If any communication exists between the pulmonary artery and the aorta—either through the canal of Botal, or by the opening of an aortic aneurism into the pulmonary artery—a loud systolic murmur will be heard in the pulmonary artery, which sometimes becomes continuous, increasing, however, in force during the systole. In the greater number of cases, a murmur heard in the pulmonary artery during the heart's systole, does not depend upon

any structural change in the artery, but upon other causes, hitherto unexplained. I have never observed a murmur in the pulmonary artery during the heart's diastole. Defect of the valves of the pulmonary artery is a very rare phenomenon. The sound is loud, and the accent falls upon it when the mitral valves are defective; when the left auriculo-ventricular opening is constricted, without valvular deficiency; when the right ventricle is hypertrophied and dilated, and the heart acts vigorously.

A double systolic sound may occur, both in the normal and abnormal condition of the heart. It merely indicates irregular action of the heart—probably a want of synchronism in the contraction of the ventricles. Neither is a double second sound any proof of structural change of the heart. It arises either from non-synchronous dilatation of the ventricles; or the two sounds are, as we before observed, the audible periods (*Momente*) of a weak murmur, attendant upon constriction of the mitral orifice, and defective aortic valves; or the reduplicated second sound (as well as the ordinary second sound), is produced by the movements of the heart.

More than two murmurs, either accompanied or not by sounds, are occasionally audible in the interval between one systole and another, in cases where endocarditis and pericarditis are both present, or where the valves are in an abnormal condition, and the pericardium roughened: the fact may be explained by the circumstance of the pericardial not being exactly

synchronous with the endocardial murmurs. Our endeavour must be to discriminate and seize upon each murmur and each sound separately; this is always possible, with the aid of the above rules, provided the movements of the heart are not very weak and rapid: the sound, or murmur, once discriminated, there is no difficulty in deciding upon its signification.

We now and then meet with cases in which two different murmurs are heard simultaneously at the same spot; such murmurs may both arise in the same part, as, for instance, in the left ventricle; or one of them may arise in the aorta, and the other in the left ventricle: they must be judged of as in other cases. The grand object is, to ascertain whence the murmur proceeds, and whether it is synchronous with the systole, or with the diastole of the heart.

IV. THE RHYTHM OF THE HEART'S MOVEMENTS.

The deviations from the normal rhythm of the heart's movements are manifold: the number of the movements, in a given time, may be abnormally increased or diminished: single movements may be unequal in duration and in force: and the relation of the length of the systole to that of the diastole altered. Several of these unnatural conditions of the heart's rhythm may be mixed up one with the other, and their combination brought under another class of irregularities; or the movements of the heart may be such as to render their reduction into any order impossible.

We form our judgment of the heart's movements, whether normal or abnormal, from the nature of its impulse and of its sounds and murmurs, and from the condition of the arterial pulse. Now, the opinion which an observer may give in any particular case respecting the character of these movements, will depend very much upon the views which he holds concerning the causes of the heart's impulse, and of its sounds and murmurs. Laennec, for instance, tells us, that in certain cases of palpitation of the heart, two or more contractions of the auricles may be observed accompanying a single contraction of the ventricles; he believed that the second sound was produced by contraction of the auricles. Bouillaud, on the other hand, speaks of an occasional double or even triple ventricular diastole, associated with a single systole.

The impulse of the heart is a sure indication of the ventricular systole; but we cannot decide with certainty upon the nature of the heart's movements by the aid of its sounds, for though we know *many*, we do not know *all* the causes upon which these sounds depend.

Abnormal rhythm of the heart's movements very frequently depends upon structural change in the heart itself; but it is nevertheless certain that the most marked irregularity in the rhythm, both of its impulse and sounds, may exist when its structure is apparently perfectly normal; on the other hand, there is scarcely a single organic alteration of the heart and its valves, which may not co-exist with the

most complete regularity in the rhythm of its movements; hence the greatest irregularity of its rhythm does not justify us in assuming the existence of organic disease of the heart.

PART II.

DESCRIPTION OF THE PHENOMENA OBTAINABLE BY THE AID OF AUSCULTATION AND PERCUSSION,

APPERTAINING TO SPECIAL CONDITIONS OF THE THORAX AND ABDOMEN.

CHAP. I.

NORMAL CONDITIONS OF THE THORACIC AND ABDOMINAL ORGANS.

The phenomena proper to special conditions of the thoracic and abdominal organs, obtainable by the aid of auscultation and percussion, are to be deduced from a consideration of the foregoing description of these phenomena. For the more full comprehension of the subject in hand, I will here give an account of the deductions which may be thus arrived at.

The percussion sound and the resistance vary much in the different regions of the thorax and abdomen, when the subjacent organs are in a normal condition. The percussion-sound also varies in different individuals over corresponding parts; and the same is true of the phenomena of auscultation.

PHENOMENA OBTAINED BY PERCUSSION.

Variations of the sound as heard in the different regions of the thorax. The right half of the anterior

surface of the thorax, between the sternum and right lateral region, and from the collar-bone down to the fifth rib, yields an equal, full, clear, non-tympanitic percussion sound, and very little resistance. From the sixth rib down to the lower border of the thorax, the sound (in consequence of the presence of the liver) is almost completely dull, resembling the percussion-sound of the thigh, and the resistance felt is considerable. Above, the dull liver-sound passes gradually into the clear lung-sound, and at the lower border of the thorax into the muffled or clear tympanitic sound of the intestines. The upper half of the sternum yields, for the most part, as full and clear a sound as the neighbouring part of the right thorax; lower down, and particularly towards the left, the sound over the sternum becomes duller, in consequence of the presence of the heart behind it; the left lobe of the liver usually reaches as far as the ensiform cartilage.

The surface of the thorax to the left of the sternum, and as far as the left lateral region, and from the clavicle down to the fourth rib, yields the same sound, and the same resistance, as the corresponding parts on the right side. The surface, from the fourth left rib to within an inch of the lower border of the thorax, or even to the border, and from the sternum to the left lateral region, offers a muffled and less full sound, and an increased resistance.

The diminution of the sound is most marked at that point where the heart comes in contact with the thoracic walls; it is also observed for the space of half an inch or more around that point. The sound.

however, in the region of the heart is not completely dull. The dulness of the sound observed beneath the heart, is partly caused by the left lobe of the liver; an inch or more above the lower border of the thorax, we may generally remark the stomach-sound, either muffled, or very clear and tympanitic; occasionally, it is accompanied by a metallic resonance — Piorry's *son humorique.* In the right lateral region, the percussion-sound is clear, but not so full as under the clavicles: from the axilla downwards, it gradually becomes emptier, and towards the sixth rib is muffled, and from thence to the border of the thorax completely dull. In the left lateral region, the sound is not so empty as on the right side. From the sixth rib downwards, we have generally the tympanitic sound of the stomach, muffled by the spleen, but becoming perfectly clear at the border of the thorax. Upwards, from the sixth rib to the axilla, percussion produces either the non-tympanitic sound of the lungs, which is at times as full as beneath the clavicles, or a combination of the lung and the stomach-sound, and louder than that heard beneath the clavicles.

The percussion-sound is much less distinct, and the resistance greater, on the posterior, than on the anterior part of the thorax. The sound is dullest, and the resistance greatest, over the scapulæ. Not less dull is the sound over the vertebræ, unless the percussion be very strong. The interscapular space, which may be increased by displacement of the scapulæ, offers a duller and emptier sound, and a greater resistance, than the parts beneath the axilla;

the dulness increasing as we proceed upwards. From beneath the scapulæ, down to the third or fourth false rib, the percussion-sound is fuller than between the scapulæ—about the sixth or seventh true rib, even fuller than in the right axilla, though less clear.

Variations of sound as observed in different individuals.—We find remarkable differences in the character of the percussion-sound of the thorax in different persons, whose thoracic organs are healthy. In one, a very gentle stroke produces a loud sound, whilst in another, and at the corresponding part, strong percussion is necessary to elicit even a moderately clear sound. The percussion-sound is incomparably louder in persons who are spare and not muscular, and whose ribs are thin and yielding, than in others of an opposite conformation. It is hardly necessary to observe, that the percussion-sound is altered by the female breast; whenever it is possible, this organ must be pressed aside, and in different directions, so as to enable us to obtain the percussion-sounds proper to the parts beneath.

The percussion-sound of the thorax is much louder in children than in grown-up persons, in consequence of the thinness of their muscles, and the flexibility of their ribs. It is also frequently louder in old, than in middle-aged individuals; a fact explained by Raciborsky, after the theory of Hourmann and Dechambre, by a supposed rarefaction of the lung-parenchyma, and by increased rigidity of the thoracic walls. My own belief is, that this loud percussion-sound in the aged, results from wasting and thinning of the muscles and ribs, and from the enlargement of the thoracic cavity, caused by the sinking of the

diaphragm; for when the muscular tissue of the aged is not wasted, it remains unaltered, and becomes as loud in the young, when the ribs and their attached muscles have become atrophied and wasted by disease, as it is in the old.

Percussion of the Abdomen.

In the normal condition of the abdominal organs, the percussion-sound of the abdomen varies, being at one time distinctly tympanitic and clear, at another indistinct, and scarcely at all tympanitic; this difference depends partly upon the varying amount of gases present in the intestines, and partly upon the pressure exerted upon the intestines by the abdominal walls; the less this pressure is, the clearer and more tympanitic will be the sound of the intestines when they contain air. The percussion-sound is not alike over every part of the abdomen; and it varies in proportion as the intestines are more readily capable of being displaced.

The loudest and clearest tympanitic sound is generally heard over the region of the stomach; it is at times accompanied by a metallic clang. The right lateral gives, for the most part, a louder sound than the left lateral region; the left lumbar region yields a completely dull sound, except when a portion of intestine happens to lie in the neighbourhood, and so occasions a tympanitic sound.

AUSCULTATORY PHENOMENA.

Auscultation of the Respiratory Organs.

When a person speaks, either no sound whatever

of his voice reaches the ear of the observer, through the thoracic walls, or merely a dull, interrupted humming is heard, from which not the faintest idea of the words spoken can be gathered. The humming is much louder in the interscapular space than elsewhere, and some of the words spoken may be occasionally distinguished there, especially at the upper part of this space—bronchophony is heard there. The bronchophony is loud, in proportion to the depth of the voice; and distinct, in proportion to its acuteness.

A loud humming, indicating the passage of the sound into bronchophony, is not unfrequently observed immediately beneath the clavicles. The humming is not heard alike over the other parts of the thorax: its force diminishes as we recede from the neighbourhood of the large bronchial tubes. Loud bronchophony is almost always audible above the clavicles, and proceeds from the larynx.

The inspiratory murmur is either vesicular, or it is indeterminate, or scarcely audible. The vesicular breathing of infants is very loud; in adults, it is most distinct in those whose muscles are weak, and whose thoracic movements are free. When the breathing is very loud, it may be even heard over those parts of the thorax beneath which there is no lung present; as, for instance, over every part of the cardiac region, and over a certain portion of the liver, and of the stomach.

In individuals whose muscular structure is highly developed, the respiratory murmur, under normal conditions, is rarely loud enough to render its vesi-

cular character manifest; in such persons, scarcely any, or only an indeterminate respiratory murmur, is audible during ordinary respiration; a deep, quick inspiration, may occasionally produce the true vesicular breathing, but this is not always the case. The loudest inspiratory murmur is heard during the deep and rapid inspirations of a person speaking, or which precede or follow coughing; but we now and then meet with healthy persons, in whom the respiratory murmur does not become distinct, even under such conditions. In the aged, it is generally indistinct, and more or less loud, in proportion as the murmur of the finer bronchial tubes and air-cells is more or less mixed up with that of the larger bronchial tubes. In old people, a peculiar modification of the respiratory murmur is occasionally met with; it is very acute, and approaches in character to a hissing sound.

When the lungs are normal, the inspiratory murmur is heard everywhere alike over the thorax; or it may be more distinct at some parts than at others, and perhaps inaudible at certain points. The vesicular murmur is purest and most distinct at those parts of the lung which are furthest from the large bronchial tubes; when very weak, it is heard loudest in the interscapular space, but generally only as an indeterminate respiratory murmur.

When the lungs are normal, scarcely any murmur is heard during expiration; it is difficult to discover a trace of even a very short and weak one; the interscapular space alone frequently produces an indeterminate murmur during expiration.

Bronchial respiration under normal conditions is

not heard except in the neighbourhood of the upper dorsal vertebræ, and not frequently even there. In cases of violent dyspnœa, however, it may be heard during expiration over the whole of the back, and even over the anterior part of the thorax, the lung being neither infiltrated nor compressed.

Auscultation of the Heart, of the Arterial Trunks, and of the Veins.

The impulse of the heart is to be sought for about the cartilages of the fifth or sixth ribs. If there be no excitement, either of the mind or body, the impulse may be barely perceptible; on the other hand, it may be so increased in a healthy heart, as to communicate a movement to the head of the auscultator: but in such case, it will be found that the shock is not communicated to so large a surface as when caused by enlargement of the heart. A concussion is occasionally observed at each systole, over the parts of the thorax which correspond to the course of the aorta and pulmonary artery, particularly in persons in whom the anterior part of the thorax is abnormally depressed towards the bodies of the vertebræ.

The sounds in the ventricles, as well as in the arteries, may be very clear and loud, or very weak and barely audible; they may resemble each other, and differ from each other; at one time the ventricular sounds are the louder, at another, the arterial. The ventricular are generally to be distinguished from the arterial sounds by this, that in the ventricular the accent falls upon the first, and in the arteries

upon the second sound. The first sound may be very loud in one or both ventricles, and the second weak or scarcely audible; whilst in the arteries both sounds may be weak, or one of them weak and the other strong; the sounds in the arteries may also differ in respect of their strength. Variations may be observed in the clearness, pitch, etc., of the sounds, both in the ventricles and in the arteries.

The second sound follows rapidly upon the first, then comes a pause, which is broken by a subsequent first sound. In some rare cases, the interval between the first and second sounds is somewhat prolonged, and the pause succeeding the second sound very short; so that it becomes difficult, or even impossible, to decide by the ear alone, which is the first and which the second sound.

Murmurs do not arise in the ventricles of a healthy heart, but in weakly individuals, whose circulation is rapid, a blowing or hissing murmur accompanying each pulse is readily excited in the large arteries of the neck, particularly in the carotid. This murmur is seldom audible in the aorta, though it may be distinctly so in the cervical arteries; when heard in the aorta, it is always very dull and weak.

In the hollow between the two heads of the sterno-cleido-mastoideus muscles, a bruit de diable is frequently audible in young persons; it takes its origin in the internal jugular vein, and is observed much more frequently on the right than on the left side. The bruit is not heard in old persons, nor in young persons when their cervical veins are distended, in consequence of obstructed pulmonary circulation. It

cannot, of itself, be considered a sign of chlorosis, or of a spanæmic condition of the blood.

Auscultation of the Gravid Uterus.

The auscultatory phenomena of the gravid uterus were pointed out by Le Jumeau de Kergaradec. He discovered the sounds of the fœtal heart, and the so-called placental murmur, already referred to. More lately, Nägele has described a bellows-sound synchronous with the pulsations of the fœtal heart. The sounds of the fœtal heart can be heard over the uterus at the sixth month of pregnancy, and become more distinct as the pregnancy advances. They are not generally audible over any considerable extent of surface, but may occasionally be heard over the greater part of the uterus; their presence is a sure sign of the fœtus being alive. No great skill in auscultation is necessary to discover the sounds of the fœtal heart, and to discriminate them from any other accidental sounds; it shews great inexperience for any one to affirm that the so-called fœtal pulse can be imitated by accidental murmurs. It was once thought that the position of the fœtus could be diagnosed from the sounds of its heart, but this idea appears to be now abandoned. It is possible, at times, to discover the presence of twins by the fœtal pulse, in those cases where the two hearts do not pulsate with equal rapidity.

We are not justified in concluding from a single examination, in which we fail to discover the pulsations of the fœtal heart, that pregnancy does not exist, or that the child is dead; but if, upon repeated

and carefully-made examinations, no fœtal pulse can be found, we are justified in concluding either that the pregnancy is not far advanced, or that the child is dead.

Whether abnormal states of the fœtal heart and extra-uterine pregnancy can be discovered by auscultation, the future must decide.

The so-called placental bruit, the mode of origin of which has been already referred to, is not nearly so valuable a sign in the diagnosis of pregnancy as the sounds of the fœtal heart; for it has been heard in cases of enlargement of the uterus and ovaries, when pregnancy did not exist. But such cases are rare, and hence the presence of this murmur over the uterus, always makes the existence of pregnancy probable.

The blowing murmur of Nägele, which is synchronous with the pulsations of the fœtal heart, must arise from some particular coiling-up of the umbilical cord.

CHAP. II.

ABNORMAL CONDITIONS OF THE THORACIC AND ABDOMINAL ORGANS.

ABNORMAL POSITION OF THESE ORGANS.

Percussion signs.—The organs of the thorax and abdomen may be otherwise perfectly normal, and perform their functions normally, and yet give rise to changes in the natural percussion sounds of these cavities, in consequence of their position being abnormal. Thus, the liver may be placed very high, and render the percussion sound of the thorax dull as far up as the right axilla; the tympanitic sound of the intestines at the same time reaching an inch, or even more, above the lower border of the thorax.

When the stomach lies high in the abdomen, and contains air, the percussion sound will be found tympanitic over the whole precordial region, as far up as the fourth rib on the left side. The liver and stomach may, on the other hand, occupy a low position, and thus produce considerable changes in the percussion sounds of the abdomen. Again, the liver may lie in the left, and the spleen in the right hypochondrium, and the heart in the right side of the thorax, etc. The presence of any portion of the intestines in the cavity of the pleura, may be generally ascertained by percussion.

Auscultatory signs.—Abnormal positions of the

liver, stomach, etc., are not indicated by auscultation; auscultation, however, assists us in diagnosing the altered position of the heart. When the liver lies high up under the thoracic walls, the respiratory murmur is not heard in the left lateral region so low down as usual; but, it should be remembered, that the murmur is generally weak in this part, when the position of the liver is normal; and, moreover, that it may be naturally weak, and almost inaudible over the whole of the thorax. If the respiratory murmur be particularly loud, it may be heard to a considerable distance downwards, even though the liver be situated high up under the thoracic walls.

ABNORMAL CONFORMATION OF THE WALLS OF THE THORAX.

Deviations from the natural form of the thorax produce changes in the percussion sound. The sound becomes louder than usual, when the ribs are flattened, and their power of resistance consequently diminished; but by arching out of the ribs, resistance is increased, and at those points where they are bent, and form projections outwards, the percussion sound is sensibly diminished. We must take these facts into consideration, in judging of the variations in the percussion sound produced by the projections and depressions met with in deformities of the sternum, and in abnormal curvatures of the ribs.

The highest degree of abnormity in form of the thorax is produced by curvatures of the spine; not only are the ribs thereby unnaturally bent, but fre-

quently one entire half of the thorax is considerably diminished in size; or the upper half of one side is much enlarged, and the lower half much diminished, the reverse of this holding good in the other half of the thorax. The abnormal form of the thorax necessarily induces a change in the position of its contained organs. In the contracted part of the thorax the lung is compressed, and in the expanded part, more distended than natural. The enlarged liver reaches high up, and stretches far to the left beneath the thoracic walls; the right side of the heart is also enlarged, and generally occupies a greater space than ordinary.

Auscultatory signs.—That portion of the lung, which is compressed and diminished in size through abnormal form of the thorax, yields either a vesicular or an indeterminate respiratory murmur, according as it is slightly or much compressed. The distended portion of lung yields a vesicular murmur, which is generally very clear, but sometimes indistinct. The murmur seldom remains clear when the abnormal form of the thorax is very considerable, for the compressed portion of lung generally produces hissing, whistling, or sonorous sounds. I have never met with a case where bronchial breathing, or bronchophony has arisen solely from pressure of the lung consequent upon spinal curvature. Curvatures of the spine almost invariably occasion hypertrophy and dilatation of the right ventricle, and, consequently, an increase of the heart's sounds over those parts of the thorax which correspond to the right ventricle; and the second sound of the

pulmonary artery is always very loud, much louder indeed than the second sound of the aorta.

ABNORMAL CONDITIONS OF THE THORACIC AND ABDOMINAL ORGANS.

I. DISEASES OF THE BRONCHIAL TUBES.

Percussion signs.—Diseases of the bronchial tubes produce no change in the percussion sound, so long as the lung-parenchyma remains healthy. In catarrhal inflammation of the bronchial tubes, in croup, in suffocative catarrh, in chronic bronchorrhœa, in bronchial hæmorrhage, and in enlargement of the tubes, the percussion sound remains the same as in the healthy lung.

Auscultatory signs.—Catarrhal inflammation of the lining-membrane of the bronchial tubes gives rise to various auscultatory signs, which signs differ according to the seat of the inflammation, whether it be in the finer or the larger bronchial tubes, or in both sets of tubes at the same time: according also to the condition of the lining-membrane of the tubes, whether it is merely swollen, or swollen and covered with secretion.

The slightest degree of swelling of the lining-membrane of the tubes renders the respiratory murmur louder and coarser than natural; thus, at the beginning of an attack of catarrhal inflammation, we often hear a very loud and coarse vesicular murmur, seated in the finer bronchial tubes; when the inflammation falls upon the larger bronchial tubes, the vesicular murmur is frequently masked by a coarse indeterminate murmur. The coarse vesicu-

lar and the indeterminate respiratory murmurs pass into the sibilant, whistling, and sonorous sounds. If the breathing is not hurried, it sometimes happens at the onset of the disease that no sounds whatever are heard over many parts of the thorax. When secretion has taken place into the finer bronchial tubes and air-cells, fine bubbling râles and hissing and whistling sounds arise; if it is not very tenacious, fine bubbing râles alone are heard.

If, in addition to the hissing, whistling, and sonorous sounds, the respiratory murmur is present, we may conclude that the secretion is scanty; but absence of the murmur when the respiration is vigorous, indicates the presence of a considerable amount of secretion. Secretions may be present in the air-cells and finer bronchial tubes, either in large or small quantity, without giving rise to murmurs; this cannot happen, however, when the quantity of secretion is small, unless the respiration is at the same time weak and languid.

The secretion in the larger bronchial tubes produces, according to its degree of tenacity, either unequal-bubbling dull râles, or, in addition to these, whistling and sonorous sounds, or these latter only. Besides these different râles, and whistling and sonorous sounds, we may also recognize respiratory murmurs, either vesicular or indeterminate. When the fluid is secreted into the larynx or trachea, the râles and sibilant and sonorous sounds thence arising are sometimes heard over every part of the thorax, the respiratory murmur, vesicular or indeterminate, either remaining audible or being completely masked by them.

The intensity of these different sounds, produced by inflammation of the lining-membrane of the air-passages, depends chiefly upon the extent and rapidity of the respiratory movements. If the respiration be slow and vigorous, large bubbling râles are produced.

Under normal circumstances expiration produces scarcely any murmur, but when the bronchial membrane is inflamed, a murmur becomes audible during expiration, presenting itself as an indeterminate murmur, or as one of the different râles, or as a hissing, whistling, or sonorous sound. Expiration may even produce a louder murmur than inspiration; and the sibilant and sonorous sounds, when they proceed from one of the large bronchial tubes, may extend to a considerable distance from their origin, and, indeed, be heard over the whole of one side of the thorax; when formed in the trachea, they may be heard of nearly equal strength over every part of the thorax. No change is produced in the thoracic voice by inflammation of the bronchial membrane.

Chronic bronchial catarrh gives rise to the same auscultatory signs as acute catarrh. The nature of these signs depends upon the degree of swelling of the bronchial membrane, and upon the quantity and tenacity of the secreted fluid.

The same is true of all diseases which produce thickening of the bronchial mucous membrane, or give rise to secretions into the air-passages: such as hooping-cough, suffocative catarrh, inflammation of the air-passages, with croupous or purulent secretion, and hæmorrhage from the bronchial tubes or air-cells.

The acute exanthemata, small-pox, measles, scarlatina, etc., abdominal typhus, pneumonia, the rapid development, and especially the softening of tubercles, are almost invariably associated with bronchial catarrh, that is, with swelling of the lining-membrane of the bronchial tubes, and with increased secretion; they offer the same auscultatory signs as catarrh of the lungs. These signs are not so constant in pericarditis, endocarditis, carditis, and pleuritis; but they generally manifest themselves in the progress of organic disease of the heart, or of long-standing pericardial or pleuritic effusions.

Enlargement of a bronchial tube may take place under a double form; the tube may be equally widened throughout its whole length, or for a portion of its length; or it may be widened, so as to form cavities of different sizes. The first kind of widening (so long as the lung-tissue surrounding the tube contains air) does not offer any auscultatory signs, other than those attending bronchial catarrh. The second form of enlargement of the bronchial tubes—the sacculated—when it pervades an entire lobe of a lung, which is adherent to the costal pleura, produces a peculiar auscultatory sign, viz., the large-bubbling dry crepitant râle — *craquement;* if the bronchial cavities are large, and the openings into them small, the craquement is preceded by a very loud hissing sound. The expiration is either free from murmurs, or is accompanied by hissing, whistling, and sonorous sounds. If a sacculated portion of the bronchus approach the surface of the lung, a pleural friction-sound is generally added to the craquement.

Enlargement and thickening — hypertrophy and ossification of the cartilages of the bronchial tubes, I have never seen developed to such an extent as to give rise to bronchial breathing, unless complete atrophy of the pulmonary substance existed at the same time. This abnormal condition of the bronchial tubes, produces the same auscultatory phenomena as catarrh, by which it is invariably accompanied.

II. DISEASES OF THE PARENCHYMA OF THE LUNGS.

Pneumonia.

Inflammation of the parenchyma of the lungs produces manifold changes, both in the percussion and in the auscultatory signs of the thorax. These changes depend upon the altered conditions of the inflamed tissue; upon the amount of the catarrhal affection, which always attends inflammation of the lungs, and upon the strength and the rapidity of the respiratory movements.

The different abnormal conditions of the lung parenchyma, effected by the inflammation, have no special auscultatory or percussion signs belonging to them. Two grand distinctions, however, may be marked in the condition of the lung, viz., whether it be permeable or impermeable to air. The lung is permeable in the first period of the inflammation, and during its resolution; and impermeable when completely hepatized.

Signs of Pneumonia, when the Lung-tissue is permeable to air, *i. e.*, at the commencement of Pneumonia and during its resolution.

Percussion signs.—The percussion sound remains

unaltered, however congested with blood the vessels of the lung may be, so long as no exudation has taken place into the lung parenchyma, and the contractility of the lung not destroyed. The truth of this is demonstrated, not only by observation of pneumonia in its first stage, but also, and more strikingly and conclusively, in cases of constriction of the mitral orifice of the heart, where the highest degree of congestion of the pulmonary vessels takes place, without producing any change in the normal percussion sound of the lungs.

An alteration of the percussion sound is first observed when effusion has taken place into the pulmonary tissue, or when the contractility of the lung is increased or diminished. In the first case, the change depends upon the relative proportion between the air in the lung-tissue and the amount of the infiltration, and is in no way connected either with the intensity of the inflammatory process or with its duration. The parts of the thorax, beneath which the infiltrated lung-tissue lies, and so long as it contains air, generally yield a somewhat tympanitic sound, provided the thoracic walls are not very rigid; the resistance offered to percussion being at the same time increased. The tympanitic sound is seldom perfectly clear; it remains full up to a certain degree (which cannot be distinctly marked) of the infiltration: when the sound becomes empty, we may be sure that hepatization of the tissue is at hand. The empty sound is caused by diminution of the contractility of the lung.

The tympanitic character of the percussion sound

continues in some rare cases, even when it has become very empty; in others again, it soon disappears, a dull, empty percussion sound alone remaining. From experiments which I have made on the dead body, I believe it is scarcely possible to recognize any changes in the percussion sound, or the resistance, unless the portion of lung inflamed be at least an inch thick, and equal in breadth to a pleximeter.

The non-infiltrated pulmonary tissue surrounding the infiltrated portion yields the normal percussion sound; if the latter be not particularly clear and full, the percussion sound of the infiltrated portion of lung may be clearer than it; in which case it is only by comparison of the percussion sound at several points, and particularly at corresponding points of the two sides, that we can determine which is the normal and which the abnormal sound.

Nothing abnormal will be observed, either in the percussion sound, or in the resistance, unless the infiltrated portion of lung-tissue lie immediately in contact with the thoracic walls. Change in the sound and the resistance, however, is possible when the whole of the inner part of the lung-tissue is infiltrated and surrounded by merely a thin layer of normal tissue.

Auscultatory signs.—The different degrees of catarrhal affection which accompanies the inflammation, and of the force and the rapidity of the respiratory movements, produce changes only in the auscultatory signs.

When the vessels of the lung are merely congested with blood, and no infiltration of its tissue nor

secretion into its air passages has taken place, the auscultatory signs are normal, or indicate merely swelling of the bronchial membrane.

Infiltration of the lung-tissue, associated with fluid secretions in the air passages, produces the same signs as bronchial catarrh, so long as air can enter into the inflamed lung. The nature of the râles heard at the commencement of, or during the resolution of pneumonia, depends upon the situation of the secretion, whether it be present in the finer bronchial tubes and air-cells alone, or in these and the larger bronchial tubes also, or in the larger bronchial tubes only; it depends, also, upon the greater or less tenacity of the fluid, and upon the respiratory movements, whether strong and rapid, or weak and slow. Hence at the commencement, and during the resolution of pneumonia, every kind of râle and hissing, whistling, and sonorous sounds may be heard, except only the consonating; the râles and sounds may be also variously combined together, and are not necessarily confined to that part of the thorax beneath which the inflamed lung lies, but are sometimes heard beyond it, and even reach over the whole of the thorax.

These different râles and sounds, may, as in simple catarrh, completely mask the respiratory murmur, whether indeterminate or vesicular; or the murmur may be heard together with them. If the respiration be weak and slow, there may be an entire absence of every kind of sound.

In some rare cases, infiltration of the lung-tissue occurs, without secretion into the air passages: and.

in still rarer cases, there is no secretion into the air passages during the resolution of pneumonia, when the infiltrated matters are being absorbed. In such cases no râles whatever are heard, and the respiration is either indeterminate or vesicular, or hissing, whistling, and sonorous sounds present themselves, and become very loud, if dyspnœa likewise exist; the vesicular breathing especially may be even stronger than that of children, and either coarser than natural, or very acute, in which case it assumes a hissing character. When the respiration is slow and weak, it sometimes happens that neither respiratory murmur, nor râles, nor hissing, etc. sounds, are audible over the inflamed lung.

Signs of Pneumonia, when the inflamed Lung-tissue is impermeable to air—Hepatization.

Percussion signs.—The thoracic walls, beneath which the hepatized lung lies, yield a dull percussion sound, and their resistance is increased, provided the hepatized portion of lung have the thickness of about an inch, and an extent greater than that of a pleximeter. The more extensive the hepatization, the duller is the sound and the greater the resistance. We must however recollect, that the flexibility of the thoracic walls varies in different persons, and that therefore the degree of dulness and resistance observed is not to be taken as an exact measure of the extent of the hepatization. When the percussion sound is empty, we may be certain that the hepatized part is of considerable thickness; and if the sound is so dull as to resemble

the thigh percussion sound, over flexible parts of the thorax, that the lung beneath is hepatized through its entire thickness.

The lung surrounding the hepatized part is either infiltrated, though still permeable to air; or it is free from infiltration, and normally distended; or it is abnormally distended, and emphysematous. Emphysema very frequently occurs around the borders of inflamed lung. The portions of lung remote from the hepatization, may be emphysematous or normal; or, in consequence of the inflammation, may be infiltrated by fluids of a consistent character, or merely by serosity,—still, however, containing air. These are the circumstances upon which the variations in the percussion sound of those parts of the thoracic walls, beneath which the non-hepatized portion of lung lies, depend.

The emphysematous lung immediately surrounding the hepatization, generally yields a tympanitic sound; but the emphysematous portion of lung remote from the hepatization, does not, as a rule, render the percussion sound tympanitic. The infiltrated parts of the lung, which are still permeable to air, also frequently yield a tympanitic sound, where they approach the thoracic walls; the healthy parts of the lung give their natural sound.

The normal percussion sound cannot be distinctly characterized, inasmuch as it varies considerably in different individuals, and at different parts of the thorax in the same individual; it is necessary therefore for us, in cases of hepatization of the lung, to compare the percussion sound, as heard at different

parts of the thorax, especially at corresponding points of the two sides, in order to discriminate the abnormal from the normal sounds; without such comparison, we can never with certainty set down any sound as abnormal, except the completely dull percussion sound, when it is heard at those parts of the thorax which, in the healthy condition of the respiratory organs, never yield such a sound.

Auscultatory signs.—When the portion of lung hepatized is extensive enough to contain within it at least one of the larger bronchial tubes, and if the tube contains air, and is not obstructed, either by solid or fluid exudations or by blood coagula; and if the communication between the air in the bronchial tube and the air in the trachea be free, then the voice of the patient will consonate in the bronchial tube, and be heard, either as loud or weak bronchophony, over those parts of the thorax which are nearest to the tube. The phenomena attending the respiration, vary according to the nature of the râles, etc., which happen to be present, either in the larynx, the trachea, or the bronchus, through which the air must pass before it enters into the bronchial tube surrounded by the hepatized tissue; the respiratory murmur may alone be heard, or, in addition to this, râles, and hissing, whistling, and sonorous sounds, or one of these râles or sounds only, or several of them combined together.

These râles, etc., may all, like the voice, consonate in the bronchial tube, which is surrounded by the hepatized tissue, and be heard at the same parts of the thorax as the bronchophony, although they take their origin at a distance.

Thus, in hepatization of the lungs, and under the conditions indicated, we may have either simple bronchial breathing, or, in addition to this, consonating râles, or consonating hissing, etc., either singly or variously combined together: the deeper and the more accelerated the respiration, the more distinct will the râles, etc., be.

Consonance of the voice, of the respiratory murmur, and of the different râles, and of the hissing, etc., sounds, arises from the same cause in each case; but when bronchophony is present, it does not necessarily follow that bronchial breathing, or that high, clear, consonating râles, etc., must be present likewise; nor, on the other hand, is bronchial breathing, etc., necessarily associated with bronchophony. The reason of this is, that not every sound consonates in the same space. Thus an indeterminate respiratory—but never a vesicular—murmur, sometimes accompanies loud bronchophony, and so also do râles, or hissing and sonorous sounds; on the other hand, bronchial respiration, or consonating râles, or the hissing and sonorous sounds, may be very distinct, when bronchophony is not present. Dull râles, etc., may also accompany bronchial respiration, and indeterminate respiratory murmurs be associated with consonating râles.

If the hepatized portion of lung be not large enough to contain one of the larger bronchial tubes; or if it be large enough, but the tube be filled with fluid or solid matters; or if the communication between the tube and the trachea be obstructed by mucus, blood, etc., then consonance cannot take

place: under such conditions, neither bronchophony, nor bronchial breathing, nor high clear râles, nor consonating whistling, hissing, and sonorous sounds, will be heard over the hepatized lung; the patient's voice will be inaudible, or recognizable only as a dull muttering: the respiratory murmur will be either indistinct, or not heard at all; and the hissing, whistling, or sonorous sounds present, not consonating, nor the râles high and clear.

Obstructions to the free communication between the bronchial tube and the trachea, caused by the presence of fluid or solid matters in the tube, may be removed by coughing, or by expectoration; thus, after coughing or expectoration, bronchophony, bronchial breathing, consonating râles, etc., frequently manifest themselves, in cases where previously they had not been heard.

The auscultatory and percussion signs are the same, whether the hepatization be red or gray, whether the lung be hard or soft, of firm or loose texture.

I have frequently examined patients suffering from pneumonia, in whose lungs newly-formed abscesses were found after death; but I have never, in any single instance, recognized the presence of abscess by the aid of auscultation or percussion. In every case, the abscess, though communicating with the bronchial tubes, was filled with pus or sanies. If the abscess be accidentally emptied of its contents, it will be rapidly filled again by the secretion, unless it be of long standing, and its walls have become solid and their secreting power diminished.

We may here observe, that there are no ausculta-

tory nor percussion signs, by which we can discriminate between inflamed lung, when it contains a small quantity of air, and when it contains no air at all; in many cases, the auscultatory or percussion signs, or both of these signs, indicate the existence of hepatization, although air still finds entrance into the inflamed lung; and it is not difficult to understand this, if we consider that consonance may be produced when only one or two large bronchial tubes are surrounded by hepatized lung of several lines in thickness, and that infiltration of a lung, which is not entirely deprived of air, may render the percussion-sound completely dull, at those parts of the thorax where it is somewhat dull under normal circumstances.

An impulse, synchronous with each beat of the heart, is not unfrequently observed over the hepatized portion of lung. Laennec believed that this was produced by propagation of the heart's impulse; but there is no doubt whatever, that the impulse felt over a hepatized lung, or a lung infiltrated by tubercle, is caused by the pulsation of the arteries passing into the lung.

Auscultation does not always yield similar signs at those parts of the thorax where the subjacent lung is not hepatized; and the reason of this is, that the non-hepatized portion of lung may be either normal or affected by infiltration in various degrees; its bronchial tubes also may be healthy or inflamed, and contain secretion; or the respiration may be hurried and deep, or slow and imperfect; and thus, at those parts of the thorax, beneath which hepatized lung does not

lic, there may be heard either a very loud or a weak vesicular murmur, or indeterminate respiration, or large- and small-bubbling non-consonating râles, or hissing, whistling, and sonorous sounds, etc. The voice is never bronchophonic.

Phenomena attending the Inflammation, when limited to a small portion of Lung.

Limited inflammation of the lungs—lobular pneumonia—does not occasion any change in the percussion sound, whether it attack one or several different parts; it is, however, almost invariably attended by the auscultatory signs of catarrh, audible either at single parts, or over the whole of the thorax.

Phenomena attending the induration which occasionally remains subsequent to an attack of Pneumonia; the cavities formed therein; and the widenings of the Bronchial Tubes.

The auscultatory and percussion signs of indurated lung, are the same as those of hepatization of the lung; and what has been said of the circumstances by which these signs are modified in hepatization, holds good in the case of induration. The presence of empty cavities in the indurated part, renders the percussion sound fuller and less dull; if the cavity is of the size of a pleximeter, and situated near the thoracic walls, it may produce an empty tympanitic sound, which becomes clearer and fuller, as the size of the cavity increases. In some rare cases, percussion produces the cracked-pot sound; the cavity must be very large to give rise to metallic tinkling.

Either bronchophony, or an indistinct muttering,

or no trace of voice whatever, is heard over those parts of the thorax which correspond to the ulcerated portion of lung; the respiratory murmur is either bronchial, or indeterminate, or altogether inaudible; and it is either clear, or mingled with every kind of râle, or with whistling and sonorous sounds: in very large cavities we may hear the metallic tinkling, or the amphoric buzzing, as the resonance of the voice, of the breathing, and of the râles. Dilatation of the bronchial tubes, in the consolidated lung which remains after pneumonia, occurs at least as frequently as the formation of cavities in it through ulceration. Dilatation of the bronchial tubes offers the same auscultatory signs as induration of the lung tissue.

The auscultatory signs of Pneumonia, as described by Laennec.

Laennec's account of the auscultatory signs of pneumonia is very different from the preceding. According to him, crepitation is a sign pathognomonic of the first stage of pneumonia; it attends the first onset of the inflammation, and is accompanied by the respiratory murmur. The extent of surface over which it is heard, corresponds to that of the inflamed lung, and frequently is not greater than the diameter of the stethoscope. Immediately in the neighbourhood of the inflamed lung, the râle becomes less distinct, and appears as though it came from a distance, and at two or three inches from it is no longer audible. As the disease advances into the stage of hepatization, the crepitating râle becomes

moister, the bubbles of unequal size, and fewer in number; at the same time, the respiratory murmur diminishes in force, and at length disappears. When the hepatization is complete, the crepitation ceases altogether.

Laennec believed that, by the aid of the crepitating râle, he could not only diagnose the more extended and superficial inflammations of the lungs, but those also which are centrically situated, and of very limited extent, even though the inflamed part should not exceed the size of an almond. Moreover, he imagined that he could determine whether or not the inflammation was centrical, and of partial extent. He asserts, that when the pneumonia is central and not extensive, the crepitation is of a deep character, and heard over a limited space, and that at the same time the respiratory murmur is clear and superficial, often, indeed, even puerile; as the inflammation approaches the surface, the respiratory murmur diminishes. He was also able to distinguish the crepitating râle, even amidst the noisy mucous râles of the dying, or of suffocative catarrh, and thereby to diagnose the existence of a central and circumscribed pneumonia.*

* Laennec seems to have very frequently observed the crepitating râle; I rarely meet with it. The reason of this difference has been accounted for, by supposing that when Laennec made his observations, pneumonia was of a different character to what it is now, being at present of a gastric type, and not pure. The division of pneumonia into pure, gastric, bilious, etc.,—although of ancient date,—is altogether based upon theory. As regards the anatomical conditions of the lungs in pneumonia, they seem to be at the present time exactly such as described by Laennec.

Absence of the respiratory murmur and of crepitation, and in many cases the appearance of bronchophony, are, according to Laennec, the auscultatory signs of hepatization of the lung. When the pneumonia is central, the bronchophony is either very indistinct, or not heard at all; in fact, it never becomes distinct, unless the inflammation approaches the surface of the lung. The bronchophony is always accompanied by bronchial respiration and bronchial cough; in central pneumonia, however, deep bronchial respiration and deep bronchial cough are present, without bronchophony; and the reason of this is, that the surface of the lung still contains air, or is simply congested, and consequently is not a good conductor of sound. If bronchial râles accompany the bronchial respiration, they will be rendered louder by the hepatization, and become audible over a greater space. The bronchophony at the roots and apices of the lungs resembles pectoriloquy, and, in observing it, we often experience a sensation, as though air were blown into the ear; the bronchophony being, in such case, accompanied by the *souffle voilé*.

So long as the inflammation is on the increase, the crepitating râle continues to be heard in the neighbourhood of the hepatized lung, or it appears at some previously healthy parts, and thus becomes, as it were, a kind of forerunner of the approaching hepatization of those parts.

The infiltration of the pulmonary tissue with pus, produces no new sign, so long as the pus remains

concrete;* when it softens down, a more or less well-marked mucous râle arises in the bronchial tubes, accompanied by cavernous respiration and pectoriloquy. If the abscess is situated near the surface of the lungs, the auscultator will experience the sensation as of air being blown into his ear (*souffle dans l'oreille*), and should some part of its walls be softened and thin, the blowing becomes converted into the *souffle voilé*. Little skill, according to Laennec, is requisite for distinguishing between the purely bronchial, and the cavernous respiration; this last evidently appears as though it proceeded from a circumscribed space, of greater magnitude than the largest bronchial tube. The intensity of the râles, added to other signs, when the abscess is half filled with fluid; the punchinello voice accompanying the pectoriloquy; the limited extent of the disease, which has been from the commencement of a partial character, or has become so subsequently to the resolution of the pneumonia in the other parts of the lungs,—are all signs which, in the greater number of cases, leave us no room for hesitation in diagnosis.

The following, we read, are the signs of the resolution of pneumonia: If the resolution commences before the pneumonia has reached the stage of hepatization, the crepitating râle gradually diminishes, and the natural respiratory or pulmonary murmur becomes more distinct, and is at last heard free from every kind of râle.

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* Fornet believes he has discovered a peculiar râle, which attends the transition of red into gray hepatization. See p. 123.

The resolution of pneumonia which has reached the stage of hepatization, is announced by the return of the crepitating râle; this is a most certain sign. I have never, observes Laennec, known it fail in any case of pneumonia which I have followed from day to day; I have given it the name of recurring crepitating râle—*râle crépitant de retour*. The murmur of the pulmonary distension becomes gradually mixed up with this râle, and is at last alone audible.

The crepitating râle, moreover, announces the commencing resolution of pneumonia which has arrived at the stage of purulent infiltration; but, in this case, it is generally preceded by a mucous or sub-mucous râle, indicating the softening down of a portion of the purulent deposit. The murmur of the pulmonary expansion is not associated with the râles at so early a period in this, as in the former case. In the course of a few days, sometimes in a few hours, the crepitating râle becomes sub-crepitant, announcing the presence of œdema, which generally attends the resolution of pneumonia in this stage. The same thing happens when œdema supervenes, during the resolution of pneumonia, in the two other stages of the disease.

Dr. Stokes speaks of a stage of pneumonia, which precedes the first stage of Laennec: he attributes the lively red colour of the lungs to the congestion of the finer arteries, and considers this to be the condition which always precedes the secretion—the crepitating râle. The rapid production of hepatization, not preceded by Laennec's signs of the first stage of pneumonia, can, according to Dr. Stokes, only occur

secondarily during typhus, but never in sthenic pneumonia. The red hepatization is caused by excessive congestion of the parenchyma, and not by deposit of lymph-exudation within it; and hence we may understand the reason of the rapid development and sudden disappearance of this morbid condition of the lungs. The rare occurrence of abscess in pneumonia, he explains by the circumstance that the disease, which is seldom circumscribed, destroys life before the abscess has time to form; he does not, however, believe that pulmonary abscess is so rare a phenomenon as it is generally thought to be. We learn from Rokitansky's *Pathological Anatomy*, that the lively red colour of the lungs is occasioned by anæmia, and has nothing whatever to do with incipient inflammation. I must reject the opinions of Dr. Stokes, respecting the nature of red hepatization, and of pulmonary abscess.

He gives us, as signs of his supposed first stage of pneumonia, the sudden advent of puerile respiration over a circumscribed spot, combined with fever and disturbance of the respiratory system. It is certainly true, that loud vesicular respiration occasionally precedes the crepitating râle; but this is no reason for setting up an especial first stage of pneumonia, this symptom being even less constant than crepitating râle.

The signs of Dr. Stokes's second stage—Laennec's first—are the presence of the crepitating râle, and the gradual disappearance of the vesicular breathing; but he lays no especial value on the crepitating râle, because it is not constantly present in pneumonia,

nor, as a physical sign, indicates anything more than the presence of secretion in the air-cells. He has not met with cases in which the completely dull percussion sound, and the bronchial respiration have passed into vesicular breathing, without any intervention of the crepitating râle.

He believes also, that the production of bronchial respiration requires not merely condensation of the lung, but also a certain degree of distension of the affected side of the thorax; and that when the entire lung is condensed, the bronchial respiration ceases, because the affected side of the thorax is immoveable: this, he considers, is the reason why bronchial respiration in pneumonia increases in force up to a certain point, then decreases, and finally, when the percussion sound is everywhere dull, and the resonance of the voice fails, ceases altogether. When resolution of the inflammation commences, even though it be over a limited space, or when an abcess forms, the bronchial respiration returns, and again for a time increases in strength.

I cannot admit these assertions of Dr. Stokes, respecting bronchial breathing: it would not be difficult to prove their untenable character from his own writing; nor can I agree with the statement, that the conversion of red hepatization into purulent infiltration is indicated by the simultaneous appearance of bronchial respiration, and of a peculiar sharp muco-crepitant râle.

It seems probable, that the kind of râle referred to by Stokes, is what I call consonating râle. He looks upon Laennec's signs of pulmonary abscess as

almost infallible, when due consideration is given to the antecedent phenomena. He explains the tympanitic percussion sound by the exhalation of gases into the pleural cavity.

Dr. J. C. B. Williams gives the following explanation of the crepitating râle: the lung-cells and finer bronchial tubes are compressed by the distended blood vessels in the first stage of pneumonia, so that the air is unable to pass through them, and the mucus, which lines them, in a continuous stream; but passes in very minute bubbles, the bursting of which constitutes the crepitation in question. The bronchophony of hepatized lung is much diminished in force when heard through a stethoscope fitted with a stopper, but this is not the case with the pectoriloquy of a cavity. The tympanitic resonance is sometimes caused by the stomach, when the left lung is hepatized; near the sternum, we may occasionally have the tubular resonance, or bottle note of the large air-tubes. In other particulars, Dr. Williams does not differ from Laennec.

I have already given my opinion of the value of the auscultatory signs, which Laennec considered as pathognomonic of pneumonia; I shall here merely refer to the order in which he places them.

In the great majority of cases, according to my experience, the auscultatory signs of pneumonia do not follow in the order assigned to them by Laennec.

It frequently happens that the fine equal-bubbling râle is not heard at the commencement of pneumonia; we meet with the unequal bubbling râle—Laennec's mucous râle—or whistling and sonorous sounds,

much more often at this period. In some rarer cases, the disease at its onset is unaccompanied by any râle at all; the inflamed parts yielding an indeterminate, or vesicular, or very noisy respiratory murmur, which passes at last into bronchial respiration.

So long as the hepatization continues, we may have bronchial breathing unmingled with râles, or combined with consonating or non-consonating hissing and sonorous sounds, or again the breathing may be indeterminate, and with or without râles, or hissing sounds, etc.; or there may be no respiratory murmur, nor any râles whatever, audible. The consonating râles may be formed of smallish bubbles, and when of a dry character resemble crepitation; or the term crepitation might rather in many instances be applied to the consonating râle. It would be hard to say, whether the râle which is heard previous to the hepatization, or the consonating râle attendant upon complete hepatization, has been most frequently taken for Laennec's crepitating râle.

The period of resolution of pneumonia does not invariably commence with the appearance of the crepitating râle; in most cases it is attended by a great variety of râles, or by whistling and sonorous sounds. In some rare cases, no râles whatever appear during this stage of the inflammation; the bronchial breathing being at first indeterminate, and at last vesicular.

The crepitating râle, or a râle resembling it, is heard during the resolution of moderately severe cases of pneumonia; it is also occasionally observed in severer cases, at a more advanced period of the

resolution, when the secretion has become scanty. In the greater number of cases, the vesicular breathing does not return immediately upon the resolution of the disease; but we generally find, after all the functions are restored to their healthy condition, and percussion no longer yields any abnormal sound, that the respiration still remains indeterminate, or that râles, or hissing, whistling, and sonorous sounds continue. Auscultation yields the same signs when the resolution is incomplete.

It follows from the above, that the presence of pneumonia cannot be determined by the auscultatory signs alone, and that these are often very indefinite, and that bronchophony, bronchial breathing, and other consonating sounds, as well as vesicular breathing, and fine equal-bubbling râles, are signs, which of themselves do not enable us to draw accurate conclusions as to the condition of the lung-parenchyma; in forming our diagnosis, we must also take into consideration every other symptom attainable by percussion, and by other means at our command.

Gangrene of the Lungs.

Gangrene of the lungs may occur as a sequel to inflammation of the lungs, or independently of pneumonia; in the latter case we find various abnormal conditions of the lungs; in the former, percussion and auscultation give us the signs of pneumonia; in the last, of catarrh, or of that particular abnormal condition which preceded the gangrene.

Laennec asserts, that the percussion and auscultation signs of gangrene are much the same as the

signs of pulmonary abscess, except that the crepitating râle is less frequently heard than in ordinary inflammation of the lungs, which may be readily accounted for by the fact, that an examination of the thorax is rarely made in the first stage of the disease, on account of its insidious nature. He has observed, moreover, that the crepitating râle first appears when destruction of the epithelial surface has taken place, and therefore indicates the existence of the inflammatory process by which it is separated. Next arise cavernous râles, and when the cavity is emptied of its contents, pectoriloquy; the pectoriloquy is of a much clearer and louder character in a gangrenous excavation, than in one formed by pulmonary abscess: and presents nothing of that flapping which appears to take place in the walls of pulmonary abscesses, and indicates the destructive process going on within them; it is also very rarely accompanied by the *souffle voilé*, so common in pulmonary abscesses. It is scarcely necessary for me to observe, that all these assertions of Laennec are arbitrary.

Laennec's Pulmonary Apoplexy (*Apoplexie pulmonaire*).

I do not think this name has been aptly chosen, for pulmonary apoplexy ought properly to indicate some anatomical change in the structure of the lungs; whereas it has hitherto been used only to point out a series of disturbances of function, without any regard to changes of organic structure. The functional disorders which occur in Laennec's pulmonary apoplexy, have no resemblance to the disturbances in the brain, produced by cerebral apoplexy. The

term signifies an effusion of blood into the parenchyma of the lungs, which is thereby rendered as dense and as heavy, as though it were hepatized; the part, on section, presents a granular appearance, and a reddish-brown colour, or a tint exactly resembling that of the venous blood.*

This abnormal condition of the lung-parenchyma is rarely met with, and very few cases of hæmoptysis depend upon it. Effusion of blood into the parenchyma has been occasionally observed unattended by hæmoptysis; the infiltrated portions of lung are rarely of sufficient size to produce any change in the percussion sound.

The auscultation of pulmonary apoplexy presents us with râles, or whistling and sonorous sounds; the infiltrated part of the lung is rarely of extent sufficient to give rise to bronchophony, bronchial breathing, etc.

According to Laennec, auscultation offers us two capital signs of hæmorrhagic engorgement of the pulmonary tissue: viz., failure of the respiration over a moderate extent of surface, and crepitating râle in the neighbourhood of the part where the respiration is not heard, being indicative of slight hæmorrhagic infiltration; this crepitating râle is only heard at the commencement of the disease, and disappears afterwards. These two signs enable us to distinguish between pulmonary apoplexy and hæ-

* According to Bochdalek, the sanguineous effusion is produced by inflammation of the finest ramifications of the pulmonary artery.

morrhage from the bronchial tubes. I have very many times sought for this crepitating râle in cases of hæmoptysis, but have rarely found it. In some of the fatal cases in which crepitation had been observed over several parts of the thorax shortly before death, no trace of pulmonary apoplexy was found. It is possible, that the crepitating râle may occasionally be associated with hæmorrhagic infiltration, but I have always hitherto observed some other kind of râle, or whistling or sonorous sounds.

Œdema of the Lungs.

Œdema of the lungs is very frequently observed after death; and there can be no doubt, that it is not in every case produced during the last moments of life, although in the greater number of cases it appears to be so. When the air is not completely forced out of the air-cells by the œdema, which it very rarely is, the percussion sound is normal, or somewhat more tympanitic than natural. Auscultation produces different kinds of râles, and hissing, whistling, and sonorous sounds, but none consonating.

In some few cases, where the post-mortem examination proved œdema to have existed some time previous to death, I observed a distinctly vesicular, and even very loud, respiratory murmur a few hours before death, unaccompanied by any râles, or whistling or sonorous sounds: the vesicular murmur, however, was of an acute character, approaching to a whistling. I have never seen an œdematous lung entirely deprived of air, unless it had been at the same time subjected to pressure.

Laennec proposes two auscultatory signs, as diagnostic of œdema of the lungs: the respiration is much less loud than what we should expect from the violence and extent of the movements attending it; at the same time, a gentle crepitation is heard, as at the commencement of pneumonia, more akin to a râle than to the natural respiratory murmur; this crepitating, or sub-crepitating râle is not of so dry a character as the râle which accompanies the first stage of pneumonia; the bubbles seem larger, and leave upon the ear a more distinct impression of moisture. Both these phenomena, mentioned by Laennec, may occur in œdema of the lungs, but they are far from being characteristic of the disease.

Emphysema of the Lungs.

Laennec divided emphysema of the lungs into the vesicular and interlobular form; in the vesicular, the air-cells are enlarged without being ruptured; in the interlobular, the air escapes from ruptured air-cells, or bronchial tubes into the cellular tissue which unites them, and produces there air-bladders of different sizes, either within the lungs, or upon their surface. The formation of an air-bladder in the parenchyma of the lungs, through rupture of an air-cell, or fine bronchial tube, can only be properly spoken of, in my opinion, when the air-bladder does not communicate with the air-cells or finer bronchial tubes, and consequently cannot expel the air contained within it.

An air bladder, even of considerable size, which communicates with a bronchial tube, may have been

formed by a distended air-cell, its walls at the same time becoming thicker. It is improbable, that air which has escaped into the cellular tissue of the lungs, through rupture of an air-cell or bronchial tube, should again communicate with the bronchial tubes. Vesicular emphysema may extend through both lungs, or through only one lung, or be confined to a lobe, or to part of a lobe, or even to single cells. In interlobular emphysema, the air-bladders may be of the size of millet seed, and even as large or larger than a goose's egg.

In vesicular emphysema, either the number of air-cells is not diminished, or they are enlarged in consequence of their number being lessened. The first case, viz. enlargement of the air-cells without diminution of their number, cannot take place throughout an entire lung, unless the thorax be abnormally enlarged; and, on the other hand, abnormal distension of the thorax always indicates emphysema, provided the distension be not caused by the presence of air, gas, fluid, or solid bodies in the pleura or pericardium. Abnormal enlargement of the thoracic cavity takes place through arching of the thoracic walls, and particularly through depression of the diaphragm. If we find, upon percussion, that the lungs extend downwards nearly to the lower border of the thorax, we may be certain that vesicular emphysema exists; and that it extends over the whole of the lung, if the percussion-sound be very full, and equally so over the whole of one side of the thorax; if the upper parts of the lung yield a less full percussion-sound, the emphysema is confined to the lower parts of the

lung. Vesicular emphysema does not render the percussion-sound tympanitic, unless the more fully inflated portion of lung is bounded by another portion of lung, that is entirely deprived of air, which not unfrequently happens in hepatization and tubercular infiltration ; or unless the emphysematous lung has completely lost its contractile power.

With the exception of these cases, emphysema of the lungs does not give rise to the tympanitic percussion-sound, whether the thoracic cavity be enlarged or diminished, or of its natural size. The percussion-sound is either very full and clear, or not more than naturally so, the difference depending in part upon variations in the condition of the thoracic walls. If the intercostal spaces are much stretched, in consequence of the distension of the thorax, its walls exhibit greater resistance and resiliency, and are more elastic than in the normal state.

Whether the vesicular emphysema is partial or general, the respiration may be vesicular or indeterminate, and free from every kind of râle, etc.: this happens when the abnormally distended lung has not lost its contractility, and when, consequently, the emphysema is of recent origin, such as we find it around hepatized portions of the lung, when their bronchial tubes are free from catarrh. But this is not ordinarily the case, for we generally (and particularly in vesicular emphysema) meet with a great variety of râles and whistling and sonorous sounds produced by the catarrh, which is almost invariably present.

Interlobular emphysema—that is, air-bladders ex-

isting in the parenchyma, or at the surface of the lungs, and having no communication with the bronchial tubes—does not produce any abnormal percussion-sound; nor is there any auscultatory sign which is peculiar to these air-bladders. Those within the lung can create no murmur, for they cause no friction, neither do those which are covered by the pleura, and lie at the surface of the lung, except when this membrane is less smooth than natural: the more elevated they are above the surface of the lung, the more readily is this friction-sound produced. When the emphysema of the lungs is not very far advanced, Laennec lays great stress upon the circumstance of the percussion-sound being very clear, whilst the respiratory murmur is very weak, or inaudible. I have never observed either the one or the other of these accidents as a constant phenomenon.

Hypertrophy of the Lungs.

The lung is said to be hypertrophied, when its walls are thickened, but the air-cells themselves not diminished in number. The air-cells of an hypertrophied lung cannot contain as much air as a healthy lung, unless the thoracic cavity be enlarged; when it is so, they may contain as much, or even more. The lung not collapsing, when the thorax is opened after death, but continuing to fill the cavity, or even to protrude beyond it, is no sign of its being hypertrophied; it only shows that there is some impediment preventing the passage of the air out of the lungs. However hypertrophied a lung may be, it

collapses in the dead body, like a normal lung, if a portion of its air can escape from the air-cells. The lung has, in itself, no power of expansion, but its contractility is continually in action; it is kept constantly distended, and so made to fill the thoracic cavity by the pressure of the atmosphere. An hypertrophied lung cannot of itself enlarge the thoracic cavity, nor an atrophied lung diminish it. The anatomical characters of hypertrophy of the lung are: increase of density, and generally, also, of firmness of parenchyma, the density not depending upon its infiltration by blood or serum, etc., and the lung still containing air. The percussion-sound of hypertrophied, is the same as that of normal lung, and differs only when the lung is much distended by emphysema. If the hypertrophy be unaccompanied by bronchial catarrh, the respiratory murmur is always very loud and vesicular.

Atrophy of the Lungs.

If the thoracic cavity be of its normal size, an atrophied will contain a greater amount of air than a normal lung, the air-cells being larger than natural. There are many degrees of atrophy of the lungs; in the most extreme, the air-cells entirely disappear, and enlarged and thickened bronchial tubes alone remain. The percussion-sound of atrophied lung is generally louder than natural; but this depends upon the simultaneous wasting and increased flexibility of the thoracic walls. The auscultatory signs are those of catarrh.

Tubercles of the Lungs.

Tubercular matter is developed in the lungs, as in other organs, in two forms: viz. as isolated granules, or as a continuous mass infiltrated through the parenchyma. The isolated tubercles may be so minute, as to escape the observation of an unpractised eye; they may cause entire portions of the lung to become impermeable to air, either through their enlarging, and so coming in contact with each other, or through the development of fresh tubercles in the intervals between them.

The percussion and auscultatory signs of tubercle are various; and the reason of this is, that the solitary tubercles are separated from each other by air-containing tissue, whilst no air can enter into the tissue infiltrated with tubercular matter, or filled up by solitary tubercles, which are enlarged and come in contact with one another. Certain special phenomena are occasionally observed to arise in the cavities, produced by softening of the tubercular matter. The quantity and quality of the secretions which are formed in the bronchial tubes and cavities (and which are rarely absent in tubercle of the lungs), have also great influence on the nature of the phenomena. Whatever the signs observed may be, we shall find that they are more or less distinct, in proportion to the greater or less extent and rapidity of the respiratory movements.

Solitary Tubercles.

Percussion signs.—Solitary tubercles do not of

themselves produce the slightest change in the percussion-sound of the lungs, even though they be scattered throughout these organs, and in very considerable quantity. Any change observed in the percussion-sound, depends upon some altered condition of the interstitial tissue, occurring in the lung thus affected; the sound is tympanitic when the tissue has lost its contractility; but the infiltration of blood, serum, etc., into the tissue, whereby the air is expelled from the lung, renders the sound dull and empty: so long as the interstitial tissue remains normal, the sound continues normal; but it is less sonorous, if the tissue be more tense and hypertrophied than natural. The statement made by Stokes, that solitary tubercles, when very abundant, produce a somewhat dull percussion-sound, is incorrect; this may be shown on the dead body.

Auscultatory signs.—The inspiratory murmur may be distinctly, and even very loudly vesicular, or it may be indistinct, or altogether inaudible, though unaccompanied by râles, or whistling or sonorous sounds. Râles of every kind, as well as whistling and sonorous, etc., sounds,—non-consonating,—may be mixed with the vesicular or indeterminate inspiratory murmur; or râles or whistling sounds alone be heard. The expiratory murmur may be altogether inaudible, or as loud and strong as that of inspiration, and, like this, be associated with râles and whistling and sonorous sounds.

Swelling of, and secretion from, the bronchial mucous membrane, soon make their appearance, when any considerable deposition of tubercle has

taken place in a lung; they may appear in the course of a few days, when the tubercles are rapidly developed, but when these are of slower growth, a longer period may elapse before they manifest their presence. In such cases, the inspiratory murmur is either vesicular or indeterminate, and the expiratory almost inaudible; in other words, the respiration may be normal.

As the deposit of the tubercular matter increases, and in many cases even at its first deposition, swelling of the bronchial mucous membrane, accompanied or not by secretion, takes place; and then the same auscultatory signs appear, as those described under the head of catarrh. The slow development of tubercles almost invariably takes place in the upper parts of the lungs, and hence, in such cases, we very frequently find the auscultatory signs of catarrh permanent there, the respiratory murmur being elsewhere healthy. Rapidly developed tubercles, however, do not manifest themselves in the first instance at the apices of the lungs, but are frequently scattered equally throughout the whole of a lung, or of one lobe. I may here remark, that there are no distinct signs by which we can with certainty diagnose the existence of acute miliary tubercles; we can only surmise their presence with a greater or lesser degree of probability. Of this fact Laennec was well aware. Dr. Stokes offers the following remarks upon the diagnosis of miliary tubercles:—"If in a case which exhibits the signs and symptoms of severe bronchitis, or in which we observe a crepitating râle, continuing without intermission, we find incomplete

dulness over a considerable extent of the surface of the thorax, unaccompanied by bronchial respiration; and if the stethoscope shows that the lung is almost everywhere permeable to air, and obstructed only at certain places, or if the crepitation be too feeble to account for the dull percussion-sound, we may diagnose the acute inflammatory development of tubercle."

According to my experience, most cases of acute tuberculosis are unaccompanied by any of these signs, and every one of them may be present, without the disease being tubercular.

Tubercles congregated together in masses, and Tubercular Infiltration.

Percussion signs.—The conglomeration of tubercles takes place in the upper parts of the lungs; at this point the tubercles are slowly developed, increase in size, and at last, by coming in contact with each other, form considerable masses.

Tubercular infiltration likewise appears in the form of a slow process of development, in the upper parts of the lungs. And hence it happens, that in by far the greater number of cases of tubercular disease of the lungs (when the disease is of some standing), the percussion-sound under one or both clavicles is duller and emptier than natural, or is completely dull; over the other parts of the thorax, the sound being normal, or louder or duller than ordinary. Generally speaking, when the sound beneath the clavicles is duller than natural in this disease, it is abnormally loud in the lateral regions of the thorax;

the reason of which is, that the lower part of the lung is more than usually distended, on account of the respiration being impeded above. Acute tubercular infiltration takes place most commonly, but not always, in the upper lobes of the lungs. It produces the same changes in the percussion-sound as hepatization.

Auscultatory signs.—So long as the tubercular mass and the tubercular infiltration are of such limited extent as not to contain within them, at least, one large bronchial tube, they do not give rise either to bronchophony, or to bronchial breathing, or to any consonating sound. Vesicular respiration may continue audible beneath the clavicles, even when tolerably large masses of tubercles are present in the upper lobes of the lungs, provided there be sufficient healthy tissue to produce it, and the bronchial mucous membrane be not swollen, nor covered by secretion. But this is not generally the case, for we almost invariably observe an indeterminate inspiratory murmur, of different degrees of strength, often indeed very strong, and in most cases accompanied by moist râles, or by hissing, whistling, and sonorous sounds; the expiratory murmur is nearly as loud, or even louder, than the inspiratory, and is likewise combined with different kinds of râles, and whistling and hissing sounds.

If the tubercular masses or infiltration be of such extent as to embrace bronchial tubes, in which the voice or the respiratory murmur can consonate, we shall hear bronchophony and bronchial breathing beneath the clavicles, provided the bronchial tubes

are not filled by any fluid or solid exudations; and should there be any râles, or whistling or sonorous sounds in the trachea, or in a large bronchial tube, we shall also hear consonating râles, or whistling and sonorous sounds. But if the bronchial tubes in question be obliterated, neither bronchophony nor bronchial breathing, nor any consonating râles, will be audible; in their place, we shall have either indeterminate respiratory murmurs, with or without dull râles, or no murmur whatever. It frequently happens, in consequence of the bronchial tubes being at one moment filled with mucus, and at another freed from it by coughing or expectoration, that in the course of a few minutes, bronchophony is heard alternating with a very dull resonance of the voice; bronchial respiration with indistinct breathing; and a clear acute râle with a deep dull râle, etc.: consonating and non-consonating sounds may be also heard at the same time.

Should the tubercular masses, or the tubercular infiltration, not be developed in the upper lobes of the lungs, the respiration beneath the clavicles may be perfectly normal; auscultatory signs of disease being presented over those parts of the thorax which correspond to the affected portions of lung.

The part of the lung which is healthy, or which contains only solitary tubercles, yields either a weak or loud vesicular or indeterminate respiratory murmur; or every variety of râles and whistling and sonorous sounds may be audible, according as the bronchial tubes are or are not free from catarrhal affections. There are no auscultatory signs, patho-

gnomonic of tubercular disease of the lung; and there are none which will enable us to determine that no tubercle is present in a lung, or in any part of it.

Tubercular Cavities.

Cavities are produced by the softening down of solitary tubercles, of conglomerated tubercles, and of tubercular infiltration; they are met with of all sizes, from a pin's head to a man's fist, and upwards. Their walls consist either of tissue infiltrated with tubercular matter, forming a more or less dense layer around them (this layer being at times of such firmness as to prevent any dilatation or contraction of the cavity), or merely of a membrane, which may be so thin as to cause the cavity to appear like a membranous sack, attached externally to the normal lung tissue. Tubercular cavities generally communicate with the bronchial tubes, and are rarely free from mucus, pus, etc. On account of these differences in their structure and contents, as well as from other circumstances, the percussion and auscultatory signs which cavities present are very varied.

Percussion signs.—When the cavity is formed within a portion of air-containing tissue, the percussion-sound remains unchanged; and this is true, not only of a small, but even of tolerably large-sized cavities. The only sound which cavities situated within healthy tissue yield, is the cracked-pot sound, but this happens only in rare cases, where the cavity approaches the walls of the thorax, contains air, and is not smaller than a pleximeter; the sound, in such

case, is more tympanitic than at other parts of the thorax.

Cavities containing air, even when deeply situated within a portion of lung infiltrated with tubercular matter, will yield a tympanitic sound, if their size be not less than that of a walnut. Several smaller cavities together, will produce the same effect. The more flexible the walls of the thorax, the more readily will the tympanitic sound be produced; the sound is clearer, the nearer the cavity approaches the surface of the lung, and fuller, the larger the cavity. The cracked-pot sound is most readily obtained from cavities which are large and superficial. According to my experience, a cavity will not yield a metallic ringing sound, unless it be of the size of a fist; but it does not necessarily yield such a sound, though it be of that size.

Auscultatory signs.—A dry large-bubbling crepitating râle is heard over large cavities, the walls of which are yielding, and which dilate and contract during inspiration and expiration, the lung being attached to the costal pleura. This râle is most readily produced when there are many cavities, of the size of a pea or a bean, scattered through the lobe; it is never heard alone, but always in combination with other râles, or whistling and sonorous sounds, in consequence of the secretion of mucus, etc., which is invariably present in the affected portion of lung. Should the other râles be very loud, this dry large-bubbling crepitation will not be heard.

When a few deep-seated cavities are present in a lung which is otherwise healthy, the vesicular

breathing, interrupted by a few bubbles of a muffled râle, may be heard over them; generally speaking, however, the murmur in such cases is not vesicular, but indeterminate. Cavities with membranous walls, situated in the midst of air-containing tissue, even though of large size, never give rise to bronchophony, bronchial breathing, or consonating râles.

Bronchophony, bronchial respiration, and consonating râles, and whistling and sonorous sounds, may take place in cavities, the walls of which have a thickness of at least several lines; and if their thickness be considerable, the breathing and the voice may be accompanied by metallic tinkling and amphoric resonance. When the walls are thick and unyielding, there is neither increase nor diminution of the size of the cavity during inspiration or expiration; no air either enters into or passes out of them, and consequently no murmurs arise within them; those that appear to proceed from them, are consonating murmurs. But murmurs may be produced within the cavities, if their walls are flexible, and permit the entrance and exit of air during respiration. Râles and whistling sounds can only arise in them, when the current of air is interrupted by mucus, etc. The movement which the secretion contained in the cavity undergoes during inspiration, and especially during coughing, is attended by râles, or by whistling sounds, when air as well as fluid is present in the cavity.

We learn from the above, that percussion and auscultation afford very few certain signs of the presence of a cavity. Experience, however, teaches us,

that tubercular masses and tubercular infiltrations do not exist for any length of time, without producing cavities; we may, therefore, safely suppose their presence, whenever the tubercular disease is of some standing. Loud bronchial breathing, loud large-bubbling râles, and bronchophony, are frequently heard over cavities; but we as often, and oftener indeed, meet with cavities which do not reveal themselves to us by auscultatory signs.

Fournet's signs of tuberculosis.

Fournet divides the first stage of phthisis into three periods.

The first period ends with the formation of the crude tubercle, and is characterized by the following features.

Altered conditions of the Inspiratory Murmur.—This is increased in force from the normal standard of ten to twelve, fifteen and eighteen. Its increase has a close relation to the progress of the tubercle. It is, at the same time, generally shortened in its duration, so as frequently to fall from ten to eight, and five. Occasionally it does not offer any remarkable deviation from the healthy standard, either in respect of intensity or duration; and is changed only, in having become of a drier, harsher, and rougher character. The dry, harsh, rough inspiration is constantly present at the commencement of tuberculosis, and gradually increases as the disease advances, until at length it disappears with the change of timbre, which takes place in the murmur.

Altered conditions of the Expiratory Murmur.—

The expiratory murmur is constantly increased in intensity and duration, from the normal standard of two, to eight, and upwards. The increase of intensity bears a pretty close ratio to the development of the tubercle. The increased intensity and duration of the expiratory murmur are constantly present, unlike many other abnormal conditions of the respiratory murmurs, which exist at one time, but are absent at another. As a general fact, the intensity, as well as the duration of the murmur is increased; the cases are rare in which the duration is alone affected. The character of roughness, harshness, and dryness is associated with the expiratory, as well as the inspiratory murmur.

Occasionally there appears a crackling of a peculiar character—*froissement pulmonaire.*

The resonance of the voice, as well as of the cough, is greater at that part where the tubercles exist, than elsewhere.

In the second period—which is an advanced degree of the first—the timbre of the respiratory murmur is altered, and the signs of the first period are all exaggerated. The lowest degree of change in timbre—the clear timbre—is first perceptible in the expiratory, and afterwards in the inspiratory murmur, a further degree of change is next observed in the expiratory, which now assumes the metallic timbre, the resonant timbre, and the blowing character, all of which, by degrees, at last attach themselves to the inspiratory murmur also. The peculiar crackling already referred to—*froissement pulmonaire*—occasionally still persists; but more gene-

rally is replaced by a dry crepitating râle—*craquement sec.* The percussion sound becomes duller, and the vibrations of the thoracic walls, if perceptible while the patient speaks, are diminished in force. Well-marked bronchophony is heard beneath the clavicle on the affected side, and the heart's sounds also are louder than natural at that point. It must not however be forgotten, that the heart's sounds are naturally louder beneath the left, than beneath the right clavicle.

The third period commences with bronchial breathing, and the conversion of the dry, into the moist crackling—*craquement humide.*

The bronchial breathing is first heard during expiration, and afterwards during inspiration.

The second stage of phthisis, is marked by the presence of moist râles having a clear timbre, as well as by a more marked degree of bronchial respiration, or by the passage of the bronchial into the cavernous breathing.

In the third stage, cavernous râles, cavernous breathing, and the cavernous voice, present themselves, and as the cavity still further enlarges, amphoric echo and metallic tinkling supervene.

Fournet offers no auscultatory sign indicative of the first stage of development of tubercles, viz., when they are yet barely visible to the unaided eye; still he thinks that he has ascertained their presence, when scattered in very small numbers, here and there, through healthy layers of pulmonary tissue forming any continuous masses.

The tubercles are more readily discovered when

superficially, than when deeply seated in the lungs; and Fournet believes that they can be distinguished when massed together in scanty groups, though when much scattered, and in very small numbers, they escape observation.

I must again repeat, that the alterations in the intensity, the duration, the dryness, the softness, etc., of the respiratory murmur, here spoken of by Fournet, as well as the *froissement pulmonaire*, and the dry and moist crackling râles, stand in no direct relation with tubercle, and depend altogether upon the catarrh, which invariably accompanies the development of tubercle, or upon accidental pleuritic inflammation, or upon altered contractility of the lung-parenchyma.

As regards the succession in which Fournet places the auscultatory signs of tubercular disease of the lungs, this much only is true, viz., that in every case where the development of the tubercle is not very rapid, the bronchial breathing and its associated signs—those belonging to tubercular infiltration, or circumscribed congregations of tubercle—are preceded by the signs of catarrh; viz., a rough, loud, vesicular inspiratory murmur, or a weak vesicular or indeterminate inspiratory murmur; a loud, prolonged expiratory murmur, dry and moist râles, varying much in the size and number of the bubbles which produce them, etc.

The statements, that the inspiration becomes shorter, and the expiration more prolonged, in proportion to the development of the tubercles: that the dry crackling—*craquement sec*—is only heard

when the tubercles are in a crude state; and that the moist crackling—*craquement humide*—indicates softening of the tubercles, etc., are all completely theoretical.

Encephaloid tumours; cysts; acephalo-cysts; earthy, cartilaginous, and chalky concretions, when present in the lungs, give rise to the same auscultatory signs, as an equal extent of tubercular matter.

III. DISEASES OF THE PLEURA.

Pleuritis.

Percussion signs.—The percussion sound is not altered by mere thickening of the pleura, nor by layers (several lines deep,) of fluid or solid exudations upon it.*

The exudation may be even an inch in depth without producing any marked alteration in the sound; so much depends upon the condition of the lung, which lies behind the exudation, and upon the flexibility or resistency of the thoracic walls. When a lung is contracted or compressed into a smaller volume than natural, and still contains air, it yields a more tympanitic, sometimes a distinctly tympanitic, and not unfrequently a louder sound, than a nor-

* Fournet says, vol. i, p. 318:—"It has been shown by post-mortem examination, that a recent false membrane, of the thickness of one line, is sufficient to produce a distinct alteration in the percussion-sound; and a diminution in the vocal vibrations of the thoracic walls has also been observed at the same time, on a level with the parts covered by the false membrane."

The value of this statement may be readily tested by experiment on the dead body.

mally-distended lung. If completely deprived of air by compression, it gives the thigh percussion sound. Percussion of a flexible part of the thorax, beneath which exudation has taken place, produces a tympanitic sound, provided the lung lying behind the exudation is not deprived of its air.

The amount of exudation which will cause the lung to yield a distinctly tympanitic sound is variable, and cannot be accurately determined. The more air, and the less solid and fluid constituents the organ contains when distended, and the greater its contractility, the smaller will the volume be to which the lung must be reduced, in order to produce a tympanitic sound, and consequently the greater the depth of the exudation; whilst, on the other hand, when the lung is infiltrated by serum, etc., and the contractile power of its tissue destroyed, a small amount of exudation suffices to bring about this change in the percussion-sound. But the greater the depth of the exudation, the duller the percussion-sound becomes, so that at last we are not able to recognize the tympanitic character of the dull percussion-sound. If the exudation take place beneath some unyielding part of the thoracic walls, a less amount of it is necessary to produce the dull character of the sound; consequently at such part a completely dull and empty percussion-sound does not necessarily indicate the presence of as large an amount of exudation, as it would if heard over a yielding part of the thoracic walls.

When the pulmonary and costal pleural surfaces are not adherent, the fluid exudation naturally gra-

vitates towards the lower and back part of the thorax, and collects there above the diaphragm; the portion of lung immediately in contact with the fluid contracts, or is more or less compressed, according to the amount of the effusion. If the portion of lung thus compressed contains air, it will continue to rise and float upon the surface of the fluid (being lighter than this), so long as the pressure of the lung above does not hinder its upward movement; but further elevation is prevented, when the upper part of the lung has itself undergone some degree of compression, in consequence of the elevation of the lower part, or when it has been rendered dense and heavy, through infiltration of blood or serum into its tissue. The portion of lung, however, once submerged beneath the fluid, is soon deprived of its air, and apparently by compression; its specific gravity is thereby increased, and it now tends to sink towards the lower part of the effusion, and does not again rise to the surface, until, either through diminution of the effusion, or through increased capacity of the thorax, the resistance offered by the upper portions of the lung to the entrance of the air, has become so great as to cause the distension and elevation of the submerged portion. By the increase of the effusion, the whole of the lung may be at last compressed, so as no longer to contain air. In this condition, it is pressed back towards that part of the vertebral column where the bronchial tubes and large vessels enter into it; and may be reduced to even less than half of its normal size, according as it contains more or less fluid or solid matters.

A lung thus completely deprived of air, after it has been for some time subjected to the pressure of pleuritic effusion, may diminish, in consequence of its blood and serum being squeezed out of it, or of its nutrition being interfered with. And hence it may, and, indeed, frequently does happen, that the fluid, which appeared for a time to fill completely the pleuritic cavity, sinks to a lower level, although its quantity is in no way diminished; the consequence of this is, that air again enters into the upper parts of the lung, and that the lung rises above the surface of the fluid. Similar results are produced by increased capacity of the thorax, either from distension of the intercostal spaces, or from greater arching of the ribs, or from depression of the diaphragm.

Partial attachments of the pulmonary to the costal pleura, at times prevent the descent of the fluid to the lowest parts of the thorax. Sacculated pleuritic exudations occur in all parts of the thorax, between the lungs and the thoracic walls, between the lungs and the diaphragm, between the lungs and the pericardium, the mediastinum, the vertebral column, and between the lobes of the lung.

The percussion-sound of the thorax may be perfectly normal, in cases where the pleuritis is attended with great pain and febrile action; and it may be strikingly abnormal, when the disease is apparently of very mild type. In most cases of pleurisy, though not in all, dulness of the percussion-sound is observed in the first instance beneath the scapulæ, and then, as the effusion increases, over the lateral region, and at length over the anterior part of the thorax; it never,

however, reaches as high before, as it does behind. The percussion-sound may be perfectly tympanitic over the upper half of the affected side, and at the same time perfectly dull over the lower half; this happens when the lower parts of the lung have been deprived of air by the effusion, and the upper portion has merely undergone slight compression.*

The affected side yields a completely dull sound throughout, when the pleura is filled by the effusion; after a time, however, the sound beneath the clavicle, and also lower down, may become clearer, and even distinctly tympanitic and full, and this without any diminution in the amount of the effusion, but in consequence of enlargement of the thoracic cavity, or of diminution in size of the lower portion of the compressed lung.

The adhesions between the lung and the parts around the effusion, which almost invariably take place, prevent the displacement of the fluid by the movements of the patient; and consequently, whatever be his position, the dull sound is heard, as a rule, over the same parts. The statement that the dull percussion-sound of pleurisy varies with the position of the patient, is in the majority of cases incorrect.

Auscultatory signs.—The thoracic voice is either not heard at all, or it is heard only as an indistinct

* Dr. C. J. B. Williams believes that, when the percussion-sound beneath the clavicles is tympanitic, the upper part of the lung is completely infiltrated or compressed. There is no difficulty whatever in coming to a correct conclusion on this subject, by the aid of a few experiments on the dead body.

humming, at those parts of the thorax which correspond to the situation of the effusion, so long as the lung is not completely deprived of air; the respiratory murmur being either vesicular or indeterminate, or altogether inaudible. The same auscultatory signs may be observed over those parts of the thorax beneath which there is no effusion, provided there be no other diseased condition affecting the lungs there. A friction-sound is produced, when the pulmonary and costal surfaces are covered by plastic exudation, and come in contact with each other, during the respiratory movements; this friction-sound is therefore a sign that, at the part where it occurs, there is no serous effusion present, and no adhesions of the pleural surfaces.

The friction sound is seldom heard at the commencement of an attack of pleurisy, probably because the freshly-formed plastic matter is not of sufficient consistency to produce it; it appears much more frequently at a later period of the disease, and particularly when absorption of some portion of the serous fluid has taken place, whereby the lung is once more permitted to come in contact with the thoracic walls. The lung is almost always covered by a layer of plastic exudation, and is not in the first instance united to the thoracic walls, so that the friction-sound may be generally produced by a strong respiratory movement.

The auscultatory signs heard over a portion of lung completely deprived of air by the effusion, are either weak bronchophony, and bronchial respiration, or one of these sounds alone; or the voice is heard,

but not increased in intensity, or it may be inaudible, and the respiratory murmur indeterminate, or likewise inaudible. The bronchial breathing and the weak bronchophony are in most cases heard between the lower angle of the scapula and the vertebræ, and somewhat above and below this line. They may also be heard over every other part of the thorax.

The bronchophony and bronchial respiration cannot take place, if the large bronchial tubes, which pass into the compressed portion of lung, are either blocked up by mucus, blood, serum, etc., or obliterated by pressure. The greater the amount of exudation, the weaker will be the consonance of the voice in the bronchial tubes, and the consonating respiratory murmur; these signs indeed may become perfectly indistinct, in consequence of the large quantity of the effusion present. Effusion which compresses only the upper portion of the lung, rarely gives rise to bronchial breathing and bronchophony, in consequence of the curved course which the bronchial tubes there take, and of their ready obliteration by pressure.

Such are the reasons, why bronchial breathing and the loud clear voice are not heard in every case of effusion, where a considerable portion of lung is deprived of air; and why, instead of these symptoms, we at times have merely an indeterminate, or no respiratory murmur at all, and either a dull weak humming when the patient speaks, or no sound whatever. At those parts of the thorax, beneath which there lies a portion of lung which is but slightly, or not at all compressed, the voice is heard

only as a dull humming, the respiratory murmur being very loud, or weak, or vesicular, or indeterminate, or almost inaudible; the last condition depending chiefly upon whether the patient breathes quickly and deeply, or slowly.*

Râles occur during pleuritis, if serum, blood, or mucus, etc., happen to be present in the bronchial tubes; hissing, whistling, and sonorous sounds arise, when the bronchial mucous membrane is swollen, or covered by any plastic exudation, or tough mucus. But râles are much less frequently present during pleuritis, than in diseases of the parenchyma of the lungs; and when the auscultatory and percussion sounds are doubtful, we may look upon the existence of a fine bubbling râle, as much more probably indicative of pneumonia than of pleurisy.

The râles heard during pleurisy present every variety, in respect of the size of the bubbles which form them, of clearness and pitch; when bronchial breathing, or the conditions necessary for its production, exist, the râles may take the consonating character, or be indistinctly audible. Râles may be heard, both at those parts of the thorax, beneath which the effusion exists, and over every other part. Whistling, hissing, and sonorous sounds are less often heard in

* Great dyspnœa may, as we have already said, cause bronchial respiration to be audible, particularly during expiration, over the whole of the back, the lung being nowhere deprived of its air. Considerable dyspnœa generally attends the formation of pleuritic effusions, and hence we have a bronchial expiratory murmur over the back, before any dulness of percussion-sound appears.

pleurisy than the râles; they occur most frequently in that portion of the lung which is not compressed by the effusion, and render the vesicular respiration inaudible.

Serous effusions in the Pleura, not occasioned by Pleurisy.—Hydrothorax.

Percussion produces a dull sound, and the dulness changes its site with change of the patient's position; in other words, the serous fluid, provided it be not fixed in any particular part by adhesions of the lung, —moves from one part of the thorax to another, as the patient moves. Similar changes of the percussion sound are produced by effusions of blood, or pus—Hæmothorax—Pyothorax—into the pleural cavity.

Pneumothorax.

By this term, we understand the presence of aeriform fluids in the cavity of the pleura; they are rarely ever found there alone, being generally attended by fluid effusions. Thus atmospheric air, when it finds its way into the pleura, in the human subject, almost invariably causes pleuritic exudation. It is probable, that the fluids,—such as the contents of tubercular cavities, the sanies of gangrene, blood, etc.,—which generally pass with the air into the pleura, assist in the production of the effusion. Gases evolved in the pleura, must proceed from the decomposition of fluid contained therein. Secretion of gas into the pleura, seems to me a highly improbable occurrence.

Percussion signs.—The percussion sound of pneu-

mothorax is, in most cases, distinctly tympanitic; but if the thoracic walls be much distended, it becomes little, or not at all so. The metallic ringing is also generally to be heard, but it is barely loud enough to reach to a distance; therefore to catch the sound, it is necessary to perform auscultation and percussion at the same time.*

The liquid which is present with the air or gas in the pleura, occupies the lowest parts of the cavity, and changes its position when that of the patient is altered; it must be of considerable amount to be discovered by percussion in cases of pneumothorax. The percussion sound is, in fact, tympanitic, even below the level of the liquid, and remains unaltered, if the depth of the fluid be not great. We may reckon the quantity of fluid present in pneumothorax as double the amount indicated by percussion.

Auscultatory signs.—Either amphoric resonance, or metallic tinkling, is associated with the voice; and during the breathing we hear the respiratory murmur, and râles, and whistling and sonorous sounds, accompanied by metallic tinkling; or there is neither metallic tinkling nor amphoric resonance audible, but only an indeterminate respiratory murmur, muffled râles, or whistling and sonorous sounds, or a dull resonance of the voice; or lastly, it may be that neither voice, nor respiratory murmur, nor râles, etc., are audible. These variations in the

* With the exception of this single case, the method of simultaneous auscultation and percussion—*i. e.* of auscultating the percussion-sound—is of no service.

auscultatory phenomena do not depend upon the circumstance of the air in the pleura being at one time in communication with the bronchial tubes, and at another not so; I have never yet met with a case of recent pneumothorax, in which the communication had become persistent; but, on the contrary, I have invariably found the opening was closed, partly by the compression of the lung, and partly by the effusion. In some very rare cases of long-standing pneumothorax, an opening into the compressed lung may be formed by ulceration, and a communication thus established between the external air, and the air in the pleura. The variations in the auscultatory signs depend upon the thickness of the layer of pulmonary tissue, which intervenes between the pleural cavity, which contains air, and the bronchial tube, where the voice, the respiratory murmurs, etc., consonate.

Tubercular and Encephaloid diseases of the Pleura.

These diseases do not cause any marked alteration in the percussion sound, unless they are of considerable extent, nor does auscultation offer us any sign characteristic of them.

IV. DISEASES OF THE PERICARDIUM.

Pericarditis.

Percussion signs.—The percussion-sound remains normal, unless a large amount of effusion has taken place into the pericardium. It is impossible to define accurately what amount of effusion is requisite to produce an appreciable change in the percussion-

sound. At one time, considerable dulness is produced by a few ounces of fluid, whilst at another, even half-a-pound and upwards will not cause any change in it. The difference is caused by the position of the lungs, which are pressed further forwards over the heart in some cases than in others.

The heart being specifically heavier than the effusion, gravitates towards the lowest part of the distended pericardium, so far at least as its attachments will permit, the fluid occupying the upper part; so that when this is not abundant, we find it collected around the base of the heart, and the origin of the aorta and the pulmonary artery, the remainder of the pericardium being occupied by the heart. There can be no exception to this, unless when, through partial or total relaxation, or bulging out of the pericardium, this membrane fails to be distended by its contents—the heart and the effusion; or unless there be some attachment between the pericardium and the aorta, the pulmonary artery, or the base of the heart.

But if the effusion is considerable, it is not confined to the base of the heart and the origin of the arteries. When the patient lies upon his back, the heart sinks in the fluid during the diastole, away from the thoracic walls; and during the systole, it is driven forwards and downwards, and thus approaches them, whilst the fluid sinks towards the back of the pericardium. It appears from this, that when effusion takes place into the pericardium, the dulness of percussion is, in the first instance, almost constantly observed about the roots of the aorta and

pulmonary artery, or in other words, in the long diameter of the heart; and that increased dulness in the broad diameter coincides with extensive effusion. There are but few exceptions to these rules. If the pericardium contain as much as two pounds of fluid, the percussion-sound becomes completely dull from the second left costal cartilage to the lower border of the thorax, and from the right edge of the sternum to the middle of the left lateral region: and the resistance also is as well marked as in cases of extensive pleuritic effusions.

Auscultatory signs.—At the commencement of pericarditis, the action of the heart is generally increased, and, consequently, its impulse stronger, and its sounds clearer than natural; but as the disease advances, the movements become weaker, often indeed they are very weak, the impulse being either not at all, or only very slightly perceptible, and the sounds of the heart and arteries very faint or inaudible. This feebleness of the heart's movements, however, may be associated as well with scanty as with abundant effusion. When there is much effusion, and the patient lies upon his back, the heart during its diastole is separated from the thoracic walls in consequence of the anterior part of the pericardium being filled by the fluid, and must, if it strike against the thoracic walls during its systole, move through a greater space than it does normally, when it remains in contact with them during its diastole.

As a rule, then, the heart's impulse is weaker than natural, when the pericardial effusion is extensive,

and its sounds are less clear and strong; both impulse and sounds may at times, also, be scarcely perceptible, or not at all so, in consequence of the heart and arteries being separated from the thoracic walls by the effusion. An hypertrophied heart may produce a distinct impulse and loud sounds, although surrounded by a considerable amount of effusion; and it must not be forgotten, that the heart's impulse is frequently very weak, and even imperceptible, and its sounds muffled or inaudible, when there is no effusion in the pericardium.

Thus the nature of the heart's impulse and sounds does not enable us to decide with certainty, either as to the presence or absence of pericardial effusion. Friction-sounds are produced by the movements of the heart, when the pericardial surfaces are covered by firm plastic exudations, and come in contact during its movements; they present themselves either as scraping, rubbing, scratching, and creaking sounds, or as bellows, rasping, or sawing murmurs.

The pericardial friction-sounds are associated with the movements of the heart, but are not exactly synchronous with its sounds; and hence we have a means of distinguishing between exocardial and endocardial murmurs. There is no difference whatever between the friction-sounds which arise within the pericardium, and those which arise on its external surface, and are caused by friction of the pericardium, during the heart's movements, against some portion of the lung, whose pleural surface is covered by exudation.

The friction-sound may be heard at the commence-

ment of pericarditis, as well as during its progress, and it may continue long after the inflammatory condition of the pericardium has subsided. It does not occur in every case of pericarditis, nor does its intensity depend upon the violence of the inflammation; neither is it a sign, that the amount of pericardial effusion is small, for it may be present when this is very abundant. If the effusion be chiefly serous, or purulent, and not plastic, no friction murmur will be audible.

Effusions in the Pericardium, not caused by Pericarditis.

Dropsy of the pericardium—*hydrops pericardii*—presents the same phenomena to auscultation and percussion, as effusion consequent on pericarditis; and this is also true of effusions of blood into the pericardium, at least so far as they have been matter of observation.*

Pneumo-pericardium.

I have never seen a case of pneumo-pericardium;

* Professor Schul had a patient under his charge in the hospital, suffering from pneumothorax of the left side, produced by violent compression of the thorax between two carriages. The movements of the heart were accompanied by a râle, which closely resembled the gurgling of a cavity—a consonating râle; and it was particularly marked during the ventricular diastole. We were of opinion, that some blood had been effused into the pericardium, and had rendered the pericardial surfaces more adhesive than natural, and that the separation of the surfaces, during the movements of the heart, gave rise to the râle in question. The patient was saved by puncture of the thorax, whereby exit was given to the confined air. He recovered completely; the pericardial murmur disappeared after a few days.

when it exists, the percussion-sound must be occasionally tympanitic. Auscultation should yield some special signs. The development of gas in the pericardium is scarcely possible, except through the decomposition of fluid contained within it. The fluid in such case would be shaken by the movements of the heart, and would give rise to a murmur, resembling that produced by fluid, when shaken in a half empty bottle.*

Adhesion between the surfaces of the Pericardium.

I am not aware that percussion and auscultation afford us any signs diagnostic of union of the pericardial surfaces. Dr. Hope tells us, that in such case, a bellows murmur is heard accompanying the first sound of the heart, and audible along the aorta, and can scarcely fail to present itself, when the heart beats strongly. But the most characteristic sign would seem to be, a sudden, tumbling (*polternd*) movement of the heart, readily discoverable by the stethoscope, and very distinct when hypertrophy and dilatation of the heart co-exist, in which case the tumbling movement generally corresponds with the systole and diastole of the ventricles. The fact is,

* In Canstatt's *Jahresbericht*, 1844, p. 216, a case—related by Brichetau, in the *Archives Générales* for March—is given, in which gas was found in the pericardium, having been evolved from a sanious effusion. Percussion produced a remarkably resonant sound; at each systole of the heart, a sound was heard resembling the stroke of a mill wheel on the surface of water. Examination after death, showed that the pericardium was distended with gas, and that it contained, moreover, 250 grammes of sanious effusion.

that neither of these signs has any relation whatever to adhesion of the pericardial surfaces. The bellows murmur arises within the heart or the aorta, through defect of the valves, or from the presence of rough deposits on the lining membrane of the ventricle or the aorta; the tumbling motion depends on irregularity in the heart's rhythm, which may occur in various abnormal conditions of this organ, and, indeed, even when it is apparently in a perfectly healthy condition.*

Tubercular disease of the Pericardium.

This abnormal condition is rarely present in a

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* [In the *Zeitschrift der K. K. Gesellschaft der Aerzte zu Wien*, April 1852, there is a paper by Skoda, on the phenomena which indicate adhesions between the heart and pericardium.

His diagnosis is based upon the signs which show that the heart's apex is drawn upwards and to the right, during the systole, and is prevented from moving downwards and to the left.

The heart's apex gives no systolic beat: it is either not perceptible, or seems to cause a shock during the diastole.

During the systole, depressions become visible in the intercostal space corresponding to the apex, and frequently in one or more spaces above it, when, in addition to adhesion of the heart and pericardium, there is likewise adhesion of the pericardium to the costal pleura.

Systolic retraction of the left intercostal spaces does not, of itself, enable us to diagnose adherent pericardium; it must likewise be shown that, simultaneously with the retraction, the heart's apex is nowhere urged against the thoracic walls.

The systolic retraction of the lower half of the sternum, is a certain sign of adhesion of the heart and pericardium, and of their fixture to the vertebral column.

The limits of the dull percussion-sound remain the same, during inspiration and expiration. See *Edinburgh Monthly Journal*, July 1852. Transl.]

degree sufficient to produce any modification in the percussion-sound. If the surfaces of the pericardium are not adherent, the tubercular disease is attended by serous effusion, and that often in large quantity. Tubercles, as well as firm plastic exudation may give rise to a friction-sound.

Encephaloid disease of the Pericardium.

This disease may, in some rare cases, be so extensive, as to produce a widely-spread dull percussion-sound.

V. ABNORMAL CONDITIONS OF THE SUBSTANCE OF THE HEART.

Hypertrophy with dilatation of both Ventricles.

The dulness of the percussion-sound is generally greater than natural over the precordial region, when both ventricles are hypertrophied and dilated; if the diaphragm, however, be placed low, the thorax abnormally widened, and the lung emphysematous, the sound may remain normal, though somewhat deeper at the lower border of the thorax.

The impulse of the heart is generally increased, and often to such an extent, that the heaving of the thoracic walls at the point where the heart comes in contact with them, is sensible both to the eye and to the touch; but this increase of impulse is not constant, for it may be weak, and even imperceptible, although the heart be hypertrophied. The sounds of the ventricles and the arteries may be very loud, weak, muffled, or inaudible; or one or more of them may be replaced by murmurs; these differences depend upon the condition of the valves, and of the lining membrane of the cavities of the heart.

Hypertrophy of the Ventricles, without dilatation.

Auscultation and percussion yield the same phenomena, in hypertrophy without, as in hypertrophy with dilatation of the ventricles, the difference being only in degree. The impulse of the heart does not impart a heaving motion to the thoracic walls, but strikes upon the ear of the auscultator, like the blow of a hammer.

Dilatation of the Ventricles without hypertrophy.

In this abnormal condition of the heart, the percussion-sound is the same as in hypertrophy with dilatation of the ventricles. The impulse does not shake the head of the auscultator, and the heaving of the thoracic walls may be slight, or even imperceptible. In pericarditis, and under all circumstances where the heart's action is much increased, the impulse of a simply dilated may become as strong as that of an hypertrophied and dilated heart. The sounds or murmurs are, as in hypertrophy with dilatation, either loud, or weak, or inaudible; they may be heard only in the precordial region, or reach over the whole of the thorax. The sounds may also be replaced by murmurs.

Hypertrophy with dilatation of the right Ventricle, the left Ventricle being normal.

The dulness of the percussion-sound is here increased in the direction of the heart's breadth; but there are exceptions, caused by particular conformations of the thorax, and especially by the position

of the lungs. The impulse is either increased, or differs but little from its normal strength. As a rule, we find the second sound of the pulmonary artery increased, and louder than the second aortic sound. Increase of its first sound is much less frequently observed. The sounds over the right ventricle are either normal or increased, or weaker than natural; over the left ventricle they are either normal or weakly, or either of them may be replaced by a murmur. The second sound over the aorta is not replaced by a murmur.

Hypertrophy with dilatation of the left Ventricle, the right Ventricle being normal,

causes increased dulness of the percussion-sound in the direction of the heart's length, except when the lungs are abnormally distended. The impulse is either increased or normal; or may be very strong at one moment and weak at another; when the hypertrophy and dilatation of the left ventricle is occasioned by defective aortic valves, the impulse may be as strong as in hypertrophy with dilatation of both ventricles. The sounds of the ventricles and of the aorta, are either normal or muffled, or inaudible, or replaced by murmurs.

Diminution of the capacity of the right Ventricle, its walls being hypertrophied, or atrophied, or normal.

When this abnormal condition of the right ventricle is coincident with hypertrophy and dilatation of the left ventricle, the percussion-sound is much the same, as in hypertrophy and dilatation of the

left, with a normal condition of the right ventricle. The heart's impulse may be increased, if the aortic valves are at the same time defective; otherwise it is weaker than natural. If the right ventricle is diminished, and the left remains of normal size, or is likewise diminished, the impulse is weak, and may be even imperceptible.

Diminution of the capacity of the left Ventricle with hypertrophy, atrophy, or normal condition of its walls.

If this state of the left ventricle is accompanied by hypertrophy and dilatation of the right ventricle, the dulness of the percussion-sound will be increased in the direction of the transverse diameter of the heart. If the tricuspid valves are defective, the impulse will be somewhat increased, otherwise it will be weaker, or imperceptible; and if the right ventricle be of normal size or diminished, the impulse will also be very feeble, or imperceptible. The second sound in the aorta is never replaced by a murmur.

Inflammation of the muscular substance of the Heart.

Neither auscultation nor percussion affords us any signs characteristic of inflammation of the substance of the heart. The percussion-sound remains normal, except when dilatation of the ventricles, or thickening of their walls, have occurred, as consequences of the inflammation; these, however, are accidents which can only happen when the inflammation has been of some duration.

The impulse of the heart is either increased, so as

to resemble that of an hypertrophied heart, or it may be weaker than natural. The sounds may be loud, or feeble, or almost inaudible; murmurs do not attend inflammation of the heart's muscular substance, unless endocarditis or pericarditis exist at the same time. The rhythm of the heart's action is altered, the rapidity of its movements increased, and its impulses of unequal force, being at one moment strong and at another weak; its sounds, also, are at one moment loud and at another feeble, etc. All these irregularities, however, occur during many other abnormal conditions of the heart, and even when the heart is apparently perfectly healthy; and they may be occasionally absent, or present only in a very slight degree, when the substance of the heart is inflamed.

Softening, or hardening of the substance of the heart—Calcareous concretions within its substance—Ossification of the coronary arteries.

There are no signs characteristic of these morbid conditions of the heart.

VI. ABNORMAL CONDITIONS OF THE ENDOCARDIUM.

Endocarditis.

In endocarditis the percussion-sound is normal, unless one or both ventricles have become dilated and hypertrophied, in consequence of the defect of the valves, etc., produced by the inflammation. Increase of the heart's size may become perceptible in the course of a few days. Its movements are generally more vigorous and rapid than usual, and at times irregular; hence the heart's impulse is increased, etc.

The sounds may be everywhere normal, or they may be louder, or duller than natural, or scarcely audible; or again, sounds may be heard at some, and murmurs at other parts. Where the current of blood is not rapid, as at the lower half of the ventricles, no murmur arises, even though the endocardial membrane be inflamed and covered by excrescences; but a rapid flow of blood over the inflamed endocardium creates a murmur which is most distinctly audible at those parts of the thoracic walls which lie nearest to the point of origin of the murmur. Inflammation may render the valves incapable of effecting perfect closure—defective—or produce constriction of the orifice at their base. In endocarditis, moreover, a systolic murmur may be audible either over the left or over the right ventricle, or be confined to the aorta, or to the pulmonary artery, the natural sounds being heard over the other parts; or the murmur may be audible over several parts at the same time.

We rarely meet with a diastolic murmur at the commencement of an attack of endocarditis; such a murmur, however, may arise in the aorta in the course of a few days, when the inflammation has rendered its valves incapable of preventing the regurgitation of the blood into the ventricle.

Defects of the Valves.

Defective mitral valves.—Here the dulness of the percussion sound is generally increased, in the direction of the heart's breadth, and this happens because hypertrophy and dilatation of the right

ventricle are the almost invariable sequels of defective mitral valves.

A systolic murmur is audible at that part of the thorax where the heart's apex strikes, and occasionally, also, to a greater or less extent, in its neighbourhood; the second sound of the pulmonary artery is increased, and the accent falls distinctly upon it. Sometimes, and particularly when the heart's movements are feeble, an indeterminate sound (*Schall*), which can neither be recognized as a true sound, nor as a murmur, is heard during the systole over the left ventricle. During the diastole, either an indeterminate sound, or none at all, is heard over the left ventricle; rarely do we meet with a tolerably clear sound there. If the deficiency of the mitral valves be considerable, the aortic sounds are weak, and, as a rule, the heart's impulse is increased.

Constriction of the mitral orifice.—The dulness of the percussion-sound is, as in the last case, increased; and hypertrophy of the heart occurs even more rapidly.

The second sound in the left ventricle is replaced by a murmur, which is often so prolonged, as to be only momentarily interrupted during the heart's systole. The second sound of the pulmonary artery is increased. During the systole, either no sound at all is heard in the left ventricle, or only an indeterminate sound; and again, a murmur may be audible there, because deficiency of the mitral valves is generally associated with constriction of the mitral orifice. As a rule, the sounds of the aorta are weak, and the heart's impulse increased, and perceptible over a greater extent than natural.

Defective tricuspid valves.—Defect of the tricuspid valves occasions distension of the right auricle, by which the dulness of the percussion-sound over it is often strikingly increased. A systolic murmur also appears in the right ventricle, the veins of the neck being simultaneously distended, and a pulsation perceptible in them; when, however, the movements of the heart are feeble, the ventricular murmur may be converted into an indeterminate sound, or be altogether inaudible. During the diastole, either an indeterminate sound, or none at all, is heard over the right ventricle; a clear sound is rarely observed.

Constriction of the right auriculo-ventricular orifice.—This abnormal condition is exceedingly rare; I have never yet observed it in the living. There are some specimens of it in the pathological museum here.

Defective aortic valves.— The dulness of the percussion-sound is almost constantly increased in the direction of the heart's length, because defect of the aortic valves invariably produces enlargement of the left ventricle, which generally assumes the form of hypertrophy with dilatation. The increased dulness of the percussion-sound, however, may be interfered with by any considerable distension of the left lung. The heart's impulse is also increased, and either a heaving of the thoracic walls, or a stroke, like that of a hammer, against the head of the auscultator, results. The second sound of the aorta is replaced by a prolonged murmur, the intensity of which is greatest over the origin of the aorta. This murmur is gene-

rally audible over a considerable extent of surface, and if the defect of the valves be considerable, the loud rushing of the refluent blood is heard at the heart's apex; a murmur is thus produced in the left ventricle instead of the second sound.

During the systole, a murmur is generally heard in the aorta, for whenever the aortic valves are defective, roughnesses are almost invariably found to exist either upon the inner surface of the aorta itself, or upon the under surface of its valves. At times, however, either a sound, or an indeterminate sound, or no sound at all, is heard during the systole. The pulse of the carotid and subclavian arteries is accompanied by a loud rasping murmur. A double murmur—*bruit de va et vient,*—is generally ascribed to deficiency of the aortic valves; it is true, that under these abnormal conditions we very frequently meet with a murmur, accompanying both the systole and diastole of the heart; but the systolic murmur is not dependent upon the defect of the aortic valves; this condition produces merely the diastolic murmur. A diastolic murmur, if it have its appropriate signs, must be considered as indicative of a defective state of the aortic valves, although there be no accompanying murmur, and a sound be heard during the heart's systole.

Constriction of the aortic opening, in consequence of faulty aortic valves.—In this case, hypertrophy and dilatation of the left ventricle is also found, though not to such an extent as when the aortic valves are defective. Percussion gives the signs of hypertrophy and dilatation of the left ventricle. The impulse of

the heart is but little, or not at all increased, provided there is no co-existing deficiency of the aortic valves.

The first aortic sound is replaced by a murmur, which generally extends to a considerable distance, and becomes indistinct only when the heart's action is feeble: when this is feeble, and the aortic valves are at the same time defective, the second aortic sound is either very weak, or very indistinct, or a murmur is substituted for it.

Deficiency of the valves of the pulmonary artery, or constriction of its orifice in consequence of faulty valves, I have never observed in the living. Such abnormal conditions are still rarer than constriction of the right auriculo-ventricular orifice.

VII. ABNORMAL CONDITIONS OF THE AORTA AND PULMONARY ARTERY, ETC.

Unevenness of the internal surface of the ascending portion of the Aorta.

The percussion-sound remains unaltered. A murmur is heard with the heart's systole, and occasionally during its diastole over the upper half of the sternum. The murmur ends in a sound, when the aortic valves close perfectly, but continues as a murmur, when they are defective.

Widening of the ascending Aorta.

The widening may be of an uniform, or of a sacculated character; its existence cannot be determined, unless the aorta comes in contact with the anterior walls of the thorax; in such case, the percussion-sound will be found completely dull at that

portion of the upper part of the sternum, behind which the aorta lies, and the resistance also increased. Besides this, the auscultator will almost invariably meet with an impulse during each systole at the part referred to, as strong, or even stronger, than that of the heart. Little or no concussion is felt over the thoracic walls, between the points where the impulse of the aorta, and that of the heart, are observed. In aneurism of the aorta, a murmur generally accompanies both the systole and diastole of the heart; but there are cases where the murmur is very indistinctly, or not at all heard, or where both sounds and murmurs exist, or the sounds only. In cases, where the thoracic aneurism has formed an external tumour, we may convince ourselves by actual contact, that both the first and the second sounds may be produced in the walls of the aneurism. A strong rasping murmur is generally heard in the carotid and subclavian arteries during the systole of the heart.

Constriction of the ascending Aorta.

Enlargement of the left ventricle generally attends this abnormal condition of the aorta, and even the right ventricle seldom fails at last to participate in the enlargement. Sounds or murmurs are heard in the aorta, accordingly as the inner surface of the constricted aorta is normal or roughened, or as the aortic valves are defective or not.

Widening of the descending Aorta.

A dull percussion-sound will not be observed over the back of the thorax, unless the aneurismal sac

be of considerable size. In two cases, where the aneurisms of the descending aorta were of very considerable size, and produced a dull percussion-sound in the back, no murmur was discovered by auscultation; but nevertheless, there can be no doubt that in aneurisms of this portion of the aorta, murmurs synchronous with the arterial pulse may be heard; such murmurs, however, do not of necessity indicate the existence of aneurism, but merely, that the inner surface of the vessel is roughened.

Aneurism of the Pulmonary Artery.

I have never observed such a condition of the pulmonary artery but in one single case:—The patient, a strongly-built man, aged forty-three, came into the hospital under my charge for œdema of the feet and ascites. According to his account, the swelling of the feet first appeared towards the latter end of November, 1841, after exposure to cold. He remained two months in a provincial hospital, and entered the General Hospital of Vienna on the 28th January, 1842. His face was the face characteristic of cyanosis, the dyspnœa great, râles and sonorous sounds were heard over the whole of the thorax, and a blowing murmur with the systole over the left ventricle of the heart; both sounds of this organ were very indistinct at its base, and a weak blowing murmur, which appeared to be propagated from the left ventricle, was observed there during the systole; the same phenomena were noticed over the right ventricle.

Percussion indicated some enlargement of the

heart, but no condensation of the lung, and no pleuritic effusion. The heart's impulse was scarcely perceptible, the pulse very small and normal in number; the urine was reduced to a few ounces in the day, of a blood-red colour, and deposited an abundant red sediment, which on examination proved to be urate of ammonia. His appetite was tolerably good, and he was somewhat thirsty. The œdema of the feet and the ascites increased, serous effusions into the pleura took place, and the man died on the 18th February, 1842. No particular change occurred in the other symptoms.

On examination after death, the trunk of the pulmonary artery was found distended to the size of a goose's egg. The orifice of the artery at the ventricle was scarcely at all enlarged, its valves healthy, and capable of perfect closure. Its two chief branches, where they sprang from the aneurism, were narrowed to the size of a crow's quill; the right ventricle was hypertrophied and dilated; the left ventricle, the aortic and tricuspid valves normal, the free borders of the mitral valves were partially thickened, but it could not be said that they were incapable of performing their function during life. The coats of the aneurism showed the same changes as are observed in aneurism of the aorta.

I need scarcely say, that in this case auscultation afforded us no signs of aneurism of the pulmonary artery; the absence of all murmur may be readily explained by the remarkable narrowing of the two chief branches of the artery.

Uniform enlargement of the pulmonary artery is

frequently observed, but I have never witnessed it of extent sufficient to produce an alteration in the percussion-sound.

Widening of the venæ cavæ, or of the pulmonary veins, never produces any change in the percussion-sound.

VIII. DISEASED CONDITIONS OF THE ABDOMINAL ORGANS.

Enlargement of the Liver.

Enlargement of the liver may depend upon hypertrophy of its substance, or upon the development within it of medullary cancer, melanosis, hydatids, or abscess. In each case, the percussion-sound is the same. An enlarged liver, if it be not depressed into the abdomen, comes into contact with a greater extent than natural of the thoracic walls, and, consequently, increases the extent of dulness of the percussion-sound in every direction over the lower half of the right side of the thorax. The percussion-sound in the left hypochondrium may be rendered duller than usual, or even completely dull, through enlargement of the left lobe of the liver. If the liver be depressed into the abdomen, the sound beneath the lower border of the ribs on the right side is perfectly dull; or the sound of the intestines behind the liver is muffled as far as the liver reaches. The degree of the resistance offered by the liver can be ascertained only through the abdominal walls.

Diminution of the Liver.

In consequence of diminution of the size of the liver, the percussion-sound may not be completely

dull over any part of the lower half of the right side of the thorax, but the natural non-tympanitic sound of the lung will be less clear immediately above the liver; over the shrunken liver itself, where it comes in contact with the thoracic walls, we recognize a muffled tympanitic sound arising from the intestines beneath. The same may happen, when the liver, without being diminished in size, has lost somewhat in thickness; such a liver may be even much larger than natural, it may reach high up into the thorax, and stretch far down into the abdomen, and yet not yield a completely dull sound at any part of its circumference, provided the intestines behind and beneath it contain air, and are not rendered tense by pressure. We are not able by percussion to discover other abnormities of the liver than those affecting its size and position.

Enlargement of the Spleen.

This is the only abnormal condition of the spleen which can be determined by percussion. Whatever be the cause of the enlargement, whether hypertrophy, medullary cancer, hydatids, etc., the percussion-sound remains the same. The spleen, when much enlarged, renders the percussion-sound dull at the lower half of the left lateral region.

Abnormal conditions of the Stomach, the Intestines, and the Peritoneum.

When the intestines are much distended by air, the percussion-sound is equally loud and drum-like over nearly the whole of the abdomen, provided the

abdominal walls be not firmly contracted; the sound is very rarely accompanied by a metallic resonance. If the abdominal walls are also much distended by the gaseous contents of the intestines, the percussion-sound over the whole abdomen is everywhere equally non-tympanitic, and less clear, than when the abdominal walls are lax.

Abdominal effusions, when not sacculated, always occupy the lowest part of the abdomen, and change their situation as the patient changes his position. The intestines containing air float upon the surface of the fluid, if permitted by the mesentery; but should the length of the mesentery prevent their doing so, then the air within them tends to collect in those portions of the intestines which are uppermost. The moveable parts of the intestines are frequently pressed upwards in a coil towards the stomach and the hypochondria. The percussion-sound of the abdomen, at the parts occupied by the fluid, is as completely dull as the thigh percussion-sound, provided there be no intestine containing air situated behind the fluid. The sound is not altered by fluid intervening between the folds of the intestines, if it does not also separate them from the abdominal walls.

In all cases of extensive peritonitis, where the intestines are much distended with air, and the abdominal walls tense, the percussion-sound is muffled, and less tympanitic than natural; and this is the case, even though the exudation be of a plastic character, and of inconsiderable thickness.

When portions of the intestines have become united together, or to the abdominal walls, they generally

contain a certain amount of air, and thus the percussion-sound over the whole abdomen becomes as loud as when the intestines are free, but it is less clear.

The percussion-sound is also frequently muffled in tubercular disease of the peritoneum; it is caused by the gaseous distension of the intestines, without which it is not observed.

Encephaloid disease of the peritoneum does not alter the percussion-sound, unless of very considerable extent: and this it rarely is, except when connected with the omentum.

Abnormal conditions of the muscular and mucous coats of the stomach and intestines frequently produce changes in the percussion-sound, by causing accumulations of air or fluids in the stomach or intestines; changes may also arise from the development of a large amount of secondary growths, as of cancer in the stomach, or of tubercular degeneration of a portion of the intestines.

The Pancreas.

This organ does not give rise to any change in the percussion-sound of the abdomen; the same is true of the mesenteric glands, even when much enlarged, and of the ureters, however highly distended.

Enlargement of the Kidneys.

The character of the percussion-sound in the lumbar region is but little affected by the size of the kidneys; it may be completely dull when the kidneys are very small, and tympanitic when they are very large. To succeed in the percussion of these

organs, the pleximeter must be firmly pressed towards the kidney, until a completely dull sound is produced, and the resistance of a solid organ felt. When such manipulation has been repeated at several spots, we may possibly arrive at some conclusions, if the kidneys have attained to a considerable size.

Aneurism of the Abdominal Aorta, Cœliac Artery, etc.

Where the aneurism comes in contact with the abdominal walls, the percussion-sound is either completely dull, or it partakes of a muffled sound, caused by the neighbouring portions of intestines. If the aneurism does not reach the abdominal walls, we must press the pleximeter down upon it, to obtain its percussion-sound.

When the walls of the abdomen are wasted, the pulsation of the abdominal aorta may generally be felt through the stethoscope, or by the fingers, and at the same time a distinct or a muffled sound may be heard; more generally, however, a bellows murmur is heard, particularly when pressure is made upon the stethoscope. In aneurism of the abdominal aorta, and of the cœliac axis, pulsation is felt over the aneurism, as well as along the aorta, and a murmur is generally heard in the aneurism, synchronous with the arterial pulse; but the blood may flow through the aneurism, without producing anything beyond a very dull and indistinct sound.

Enlargement of the Uterus and Ovaries.

The uterus, on account of its deep position in the pelvis, and the ovaries, from the smallness of their

size, when normal, do not affect the percussion-sound. When the pregnant uterus rises out of the pelvis, or the ovaries are enlarged, the percussion-sound over them is dull, wherever these organs come in contact with the abdominal walls.

Distension of the Bladder, and its enlargement from thickening of its coats.

When the bladder is distended with urine, blood, etc., the percussion-sound above the pelvis is dull; it is so also when the bladder is enlarged, through thickening of its walls from hypertrophy, or from any other abnormal change. In this latter case, however, the resistance felt is greater than when the bladder is distended by urine.

Calculus in the Bladder.

The blow or the friction of a catheter upon a stone contained in the bladder, is heard much more loud and distinct, through a stethoscope placed upon the pubes, than through the air, even when the ear of the observer is brought very near to the pubic region. If the bladder contain no stone, the movement of the catheter within it gives rise to an occasional gurgle, or to a dull kind of murmur, which has no resemblance whatever to the blow produced by the striking of the catheter against a stone. Auscultation must be considered of some value in the diagnosis of urinary calculi. It aids and supports the sense of touch.

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